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ON THE SYMPTOM-COMPLEXES OF LETHARGIC ENCEPHALITIS WITH SPECIAL REFERENCE TO INVOLUNTARY MUSCULAR CONTRACTIONS.

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I.—INTRODUCTION.

WHEN a disease, particularly one involving the central nervous system, is isolated in the first place by clinical observation, it follows of necessity that only cases corresponding to a single symptom-complex can be identified as belonging to it. With increasing knowledge the originally clear-out conception of the malady becomes inadequate to include the inevitable variations from type that are observed. It has to be broadened and it loses the artificial precision of its earliest form.

Investigations into the ætiology and morbid anatomy of the malady proceed simultaneously with the extension of knowledge on the clinical side, and, finally, it becomes possible with reasonable accuracy to describe a clinical picture characteristic of the disease and to establish its main clinical variations and forms. It may be possible in some cases to correlate these with variations in the intensity or distribution of the underlying lesions in the nervous system.

It is clear that during this phase of growing experience much modification and revision of opinion as to the significance and value of observed phenomena may be necessary.

In the case of lethargic encephalitis we are still in this phase of growing knowledge. We know little of its ætiology and the full range of its clinical manifestations has probably not yet been determined. In the absence of any bacteriological or serological standards it is not always possible to decide whether a given symptom-complex should be included in the rapidly growing group attributed to this disease or not. It seems probable from a study of the literature that from time to time cases are incorrectly included, while others that belong to the group may equally well be rejected from lack of these standards.

The original clinical picture by which, under the name of botulism, the disease was first diagnosed in this country, soon gave place to various forms or types, the number of which is steadily on the increase. As Buzzard and Greenfield [5] have pointed out, it is to be expected that an inflammatory disease of the nervous system should give rise to a versatile symptomatology; the structure of this system is so complex and the functions of its several parts so highly specialized. Indeed, it is easy to conceive of innumerable possible combinations of symptoms—that is of possible syndromes—we might reasonably expect to encounter, though it would not be correct to call them all "types."

Many schemes of classification for the various syndromes of lethargic encephalitis are to be found in the literature. Most of them are purely symptomatic; some prominent symptom in a series of cases being chosen as the basis for the definition of a type. Other writers employ mixed anatomical and symptomatic groupings. The result has been that each case either simultaneously or at some stage of its course falls into almost as many groups as it has symptoms. The recently named "myoclonic encephalitis" affords a good example of this. Rhythmical clonic contractions and fibrillary twitchings of muscles, or of parts of muscles, have been recorded in lethargic encephalitis by numerous observers during the past two years. In the recrudescence of the disease during the past winter (1919-20) they have been a remarkably prominent feature. In some recently reported cases they have dominated the clinical picture entirely (Ellis, [10]), while in a few instances (Sicard and Kudelski [17, 18, 19]) clonic muscular contractions have been the only somatic nervous symptom observed. At first sight it might seem justifiable, with Sicard, to regard so clear-cut and striking a symptom-complex as a new and distinct type of the disease. However, experience indicates that the particular combination of symptoms recorded by Sicard is extremely rare in a pure form; that usually it occurs either

superimposed upon, preceding, or following a whole series of other symptoms in any given case; and that clonic muscular contractions occur as a symptom in cases of every clinical variety. Nevertheless, following Sicard, numerous writers now group as "myoclonic encephalitis" cases differing in the widest possible manner and often having nothing in common clinically except clonic twitchings of one or more muscles or parts of muscles. Moreover, the term "myoclonic" has been applied to such a variety of involuntary muscular contractions and movements by French writers that it has come to have little or no meaning. The unsatisfactory nature of classification of this kind is exemplified by such a case as that reported by Bourges and Marcandier [4] as a "forme mixte à symptômes intriqués (co-existence de somnolence, paralysies partielles oculaires, mouvements choréo-athétosique, secousses myocloniques, catatonie et troubles délirants"). While Kahn reports [12], "Un cas d'encéphalite successivement névralgique, psychique, choréiforme, myoclonique, léthargique avec séquelles hémimyoclonique après la guérison."

Such complex syndromes are by no means exceptional and the disease which can produce them clearly defies symptomatic classification.

Quite possibly no permanent classification can be devised in the present incomplete state of knowledge, but it is reasonable to suppose that an analysis of the features of the disease might reveal some fundamental character upon which a uniform scheme of description could be founded.

The symptoms of lethargic encephalitis fall naturally into the following groups:—

General Symptoms—Signs of toxic infection ..			Fever. Shivering. Cutaneous eruptions. Pains and malaise. Headache. Gastro-intestinal disorders, such as vomiting, constipation, foul tongue, &c.
Nervous Symptoms	General	"Positive"	Delirium, mania, restlessness, hallucinations, &c.
		"Negative" ..	Lethargy, coma.
	Focal ..	"Positive"	Convulsions. Involuntary movements and muscular contractions Ataxy, rigidity. Pains.
		"Negative" ..	Paralyses.

The term "positive" and "negative" are employed in the sense in which Hughlings Jackson originally used them—"positive" symptoms being expressions of exalted function either from irritation or from loss of higher control, and "negative" symptoms expressing depression or loss of function from destruction of nervous structures or otherwise. A recognition of these two classes of symptoms is essential to a comprehension of any nervous disease.

Not all these symptoms are represented in a single case, nor are those present of equal value in every instance. Thus, the general symptoms of toxic infection are minimal or entirely escape observation in some instances, while in others they dominate the clinical picture at the onset or even throughout the illness.

If ten consecutive cases were taken and considered in the light of the scheme given above, it is quite possible that not a single symptom would be common to them all. If we seek for a common factor we shall find it only in the presence of focal or localizing nervous symptoms.

Among those symptoms that are more definitely focal in origin, the localization of the positive motor phenomena presents considerable difficulty, and very diverse views have been expressed as to their cause and site of origin. Therefore, as they may be the only focal symptoms present in a given case, an attempt to analyse their nature and cause is an essential preliminary to any sound basis of description or classification. This question will be considered later, but in the meantime a few general observations on the symptomatology of lethargic encephalitis may be made.

At the time of its first recognition, the disease was remarkably "negative" in the character of its symptoms: lethargy and paralyses of certain motor cranial nerves made up its clinical picture in most instances, and although certain "positive" general and focal nervous manifestations were recorded from 1918 onwards, they were certainly not so characteristic of the malady as they have since become. Doubtless, there are many factors responsible for this apparent change in the clinical aspect of lethargic encephalitis during the past few months. It is very easy to exaggerate the importance of this change. Undoubtedly a widened conception of the disease and the admission of cases that could not have been recognized earlier as belonging to it have been contributory causes. But it also must be remembered that although overlaid by more obtrusive positive symptoms, the same negative symptoms that attracted attention to the disease originally still occur with considerable frequency. As Netter [15] has pointed

out in this connection, it has long been known that a poison may cause either exaltation or depression of function, at one time producing convulsions, at another paralysis, as alcohol, lead, and the virus of rabies are well known to do. Further, the clinical variations of an infective disease in successive epidemics are quite familiar, and although there certainly is some evidence of more extensive involvement of the nervous system now than in the earlier cases, yet we need not regard a change in the localization of the virus as even the most important of the many factors concerned.

Whatever their cause may be, the frequency and variety of "positive" symptoms, both general and focal, have increased the clinical diversity of lethargic encephalitis still further, so that a comprehensive view of its essential features is becoming increasingly difficult.

Nevertheless, an appreciation of the fact that the same principles govern the localizing of "positive" as of "negative" symptoms, of a clonic contraction as of a palsy, renders the problem less complex than appears at first sight and makes the prospect of finding a satisfactory uniform basis of description more hopeful.

A consideration of *acute poliomyelitis* in the light of the scheme given above offers many interesting points of contrast between that disease and lethargic encephalitis. In the former the general symptoms are far more constant in form and duration than in lethargic encephalitis. They always precede the onset of paralysis and rarely last more than four or five days. When they abate the activity of the virus may be considered to be spent. In lethargic encephalitis, on the other hand, as we have seen, they vary greatly in intensity, form and duration, and, as Netter [15] has suggested, it is probable that the virus remains active for weeks or months. The nervous symptomatology of poliomyelitis is characteristically "negative" and predominantly focal in type. Yet even in this disease, when the cerebrum is involved, convulsions may occur. In this connection a case described by Leonard Parsons and quoted by Batten [3] as one of poliomyelitis is of considerable interest at the moment. It was that of a child of 2, who was suddenly taken ill with fever, a fit and sleepiness. The fits recurred next day; the child regained consciousness but remained for three weeks "apparently conscious, took her milk, but her eyes were fixed and staring, and she did not appear to see anything nor did she speak." For three weeks her limbs showed a slow rhythmic tremor and were rigid. There was no paresis nor any change in the

reflexes. Recovery ensued after five weeks. In 1913 this case was diagnosed as poliomyelitis, it is doubtful if it would be so called now. With rare and questionable exceptions such as this, it may be said that the nervous symptoms of poliomyelitis are negative, or paralytic, and do not show the selective and restricted character of the palsies seen in lethargic encephalitis, which commonly involve certain motor cranial nerves only. For the long-continued fibrillary and clonic contractions of muscles seen in the latter disease there is no parallel in poliomyelitis.

In poliomyelitis classification and description have an anatomical basis. The disease is much less polymorphic than lethargic encephalitis, and its focal symptoms are readily interpreted. Yet even here Wickmann found it necessary to add an "abortive" group of cases to his purely anatomical grouping. There is no reason why in the case of lethargic encephalitis a similar uniform basis of description should not be attempted, though, undoubtedly, the problem is far more complex. Nevertheless, nothing but good can come from the abandonment of the numerous symptomatic classifications that serve only to obscure our outlook on the disease.

II.—Clinical Cases.

In this section are briefly described seven cases which were observed successively in the practice of University College Hospital. Five of them were under the care of the Medical unit, and for permission to record the remaining two I am indebted to the courtesy of Sir John Rose Bradford (Case 1) and Dr. F. J. Poynton (Case 5).

These cases were not selected, and their clinical diversity is not, therefore, artificial, but represents the disease as it has been observed in the practice of a single hospital during the past few months, though the cases are not recorded in the order in which they were seen.

At the head of each case the symptoms are summarized according to the scheme given in the first section of this paper.

Case 1.—*Lethargy and Signs of a Cortical Lesion.*

General Symptoms.—Fever, breathlessness on exertion, anorexia, constipation, foul tongue, congested fauces, headache and diffuse pains.

Nervous Symptoms. — (General) Lethargy passing into coma, general muscular hypotonus and diminished tendon reflexes. (Focal) Right hemiparesis and Jacksonian fits.

Alice R., married, aged 42. On January 12, 1920, she suddenly came over

faint and giddy while at housework. She lay on her bed and went to sleep. During the following week she complained of headache, lassitude, vague, fleeting pains in limbs and body, and increasing drowsiness, so that at times she could hardly keep awake. She was very breathless on exertion. For about a week she kept at her work, and her husband appeared to notice nothing abnormal in her. She had previously enjoyed excellent health.

On January 19, 1920, she suddenly became very much worse; she noticed that she was weak on the right side, and that her right arm and leg were twitching.

On examination on the following day she was very somnolent and slept unless aroused. This was easily done, however, and she could then give a rational account of her illness. Temperature was 103° F., pulse 132, and respirations 32. Her tongue and lips were dry and coated and the fauces congested. The thoracic and abdominal viscera appeared healthy. The urine was normal. She complained of headache and of pain in the right side of the chest.

At increasingly frequent intervals, until her death on February 9, 1920, there were right-sided Jacksonian fits of face, arm, hand and leg. The clonic jerkings began in the thumb and index-finger and spread up the arm. The lower part of the face on that side and the leg were simultaneously involved. The movements, which occurred at the rate of about three in two seconds, were most forceful and enduring in the hand and forearm; the leg was least affected. In the upper limb the flexor group seemed most affected; in the leg only the limb extensors and plantar flexors could be felt to contract. Associated with this condition was a progressive right hemiparesis, in order of severity, hand, arm, face, leg. The muscles were flaccid. All tendon jerks were sluggish, right and left equally. No abdominal reflexes were obtained, and the right plantar response was extensor, the left normal. As far as could be determined there was no disturbance of cutaneous sensibility; other forms could not be tested. There was incontinence of urine and fæces.

The patient gradually became comatose, broncho-pneumonia developed, and she died four weeks from the onset of the illness. At no time were there delirium, pain, or ocular paralyses. There was no increase of lymphocytes in the cerebrospinal fluid.

At post-mortem the soft meninges appeared congested, but no other abnormality was seen. The precentral and post-central gyri and the basal ganglia of both sides, the cerebellum, midbrain, and medulla were examined microscopically. In the cerebral cortex and the basal ganglia there was considerable round-celled infiltration of the perivascular spaces, but no other lesion was noted. No difference could be detected between the two sides. The cerebellum and brain-stem were normal.

In this case the focal symptoms indicated a combined irritative and destructive lesion of the cerebral cortex; the paresis and the convulsions having the same localizing value.

Case 2.—Lethargy and Signs of Basal Ganglia and Tegmental Lesions.

General symptoms.—Constipation, fleeting limb pains.

Nervous symptoms.—(General) Lethargy. (Focal) Defective upward movement of eyes, nystagmus, mask-like face, rigidity and tremor of left limbs.

Harry T., aged 14. His mother stated that five weeks before his admission to hospital he came home from work on a Saturday afternoon complaining of being very sleepy. He lay on his bed and slept with intervals, when he was waked for meals, until Monday morning, when his drowsiness was still unabated. During the succeeding five weeks he went to sleep whenever he was left to himself, but was always easy to rouse. He ate his meals fairly well and seemed "right in his mind," though his mother noticed that he "grinned silly" at her when spoken to, and that his left arm and hand shook and were clumsy when in use. He was very constipated, and complained from time to time of vague pains in his limbs. These were never severe.

On examination (July 5, 1920) he lay in bed apparently asleep. He awoke and became quite alert when addressed and could answer simple questions readily and accurately. He displayed a complete loss of initiative, and neither moved nor spoke unless made to do so. His face was vacant and expressionless, but periodically a slow smile dawned on his face, spread, and remained fixed often for as long as a minute before it faded slowly away. Articulation was normal, and he could swallow normally. Vision and hearing normal.

The pupils were normal. Upward movement of the eyes was distinctly limited in range. Lateral movements were normal in range and association. On fixation, particularly on lateral deviation, a coarse, rapid nystagmus appeared. This was better sustained and more rapid on looking to the left than on looking to the right. No vertical nystagmus, no squint or diplopia. No ptosis.

There was moderate paresis of both halves of the face, especially of the upper part; thus the frontales were extremely weak. In movements carried out to order the right half of the face acted more rapidly and amply, but on smiling the left half "took up" much sooner and acted better than the right. The other cranial nerves showed no defect.

Motor system: The left arm and hand were held flexed and abducted, but otherwise his general attitude was in no way peculiar. All mass movements were carried out very slowly as though he were stiff or sore. He moved and walked just like a case of paralysis agitans, and retropulsion was easily induced by making him hold back his head or by a slight push. The left arm and leg showed a curious "sticky" rigidity like that seen in paralysis agitans, and clearly differing from that seen in lesions of the pyramidal system. Movements of the left hand and digits were very slow and clumsy and a fine tremor of the forearm and fingers appeared on movement. The leg also was slow in movement but there was no tremor. Isolated movements of the right limbs were carried out with normal facility and speed, but in mass movements the pace seemed to be set by the capacity of the affected left limbs.

There was no weakness of the left limbs, power and muscular development were normal throughout. The tendon jerks were of normal facility and equal on the two sides. The abdominal reflexes were all present and the plantar reflexes flexor in type. Sensation was normal and there was no pain. Sphincter control was normal.

There was no fever. The cerebrospinal fluid was normal in every respect, there was no lymphocytosis or increase of globulin. Wassermann negative.

During the following four weeks the condition altered very little, but the lethargy gradually lessened.

At present (September 1, 1920) there is no lethargy, the rigidity and slowness of movement of the left arm are improved, but general movements, the defect of eye movements, the facies and nystagmus are unaltered.

In this case the focal symptoms indicate a lesion of the basal ganglia and the tegmental region of the midbrain. The unilateral character of certain of the symptoms is unusual, but in other respects they are of a kind quite familiar and may be regarded as examples of a mechanism released by defective activity of co-operative centres.

Case 3.—Delirium and Extreme Restlessness (Choreiform) followed by Lethargy. Signs of Midbrain (peri-aqueductal) Lesion.

General symptoms.—Fever, breathlessness on exertion, anorexia, constipation, foul dry tongue and lips, morbilliform eruption and joint pains.

Nervous symptoms.—(General) Insomnia, nocturnal delirium, hallucinations, extreme restlessness resembling severe chorea. All passing suddenly into profound lethargy after thirty-six hours. General muscular hypotonus and diminished tendon jerks. (Focal) Third, fourth, sixth and seventh nerve palsies.

Louisa F., aged 14. On May 24, 1920, the patient's mother noticed that she was very short of breath on exertion. Two nights later she began to be sleepless and restless at night. This became nightly worse, so that on the night before admission she was violent, could scarcely be kept in bed, threw herself about, appeared to be having terrifying visions and shouted that men were after her. At the same time "a rash suddenly came out on her like measles." During this period of onset she was constipated, had no appetite, complained of severe pain in the right wrist which made her cry out at times, and she seemed feverish. Admitted to hospital as a case of chorea.

On admission (May 31, 1920): She lies on her back in bed, continuously tossing about. She flings her head and limbs about with great force and has to be kept from falling out of bed by a strong sheet. The condition resembles severe adult chorea. She is flushed, her skin is hot and dry, her lips are dry and cracked and her tongue coated. Temperature 102° F.

Over legs, elbows, buttocks and shoulders there is a bright red papular rash. At the apex of the papules are small dark red crusts like dried hæmorrhages.

Although this is most marked over areas where friction is greatest it is also present on protected areas of skin and cannot be entirely due to rubbing.

Nervous system: She is in a muttering stupor when not disturbed, but when spoken to half opens her eyes and gives rational replies to simple questions, though she can give no account of her illness. Her answers are perfectly articulated.

Vision and hearing appear unaffected. She does not complain of tinnitus, but says that she sees double.

Cranial nerves.—Her pupils are equal, central and circular. They react fairly well to both light and accommodation. There is marked double ptosis with feeble overaction of the frontales when she opens her eyes. The eyes do not move at all parallel, but no single muscle can be incriminated and all movements appear limited in range. There is no nystagmus. There is diplopia. There is profound weakness of both sides of the face. She can close her eyes, but there is scarcely any movement round the mouth. Her tongue is protruded straight and well and can be maintained steadily protruded. All its movements are perfectly normal. The motor and sensory fifth nerves are normal. Articulation, deglutition and phonation are normal.

Motor system.—There is general hypotonus and some general weakness, but no true paresis, nor localized weakness. The constant movements of head, trunk and limbs closely resemble those of chorea, but there appears some evidence of voluntary control when carrying out movements to order. The steadiness of the tongue moreover is unusual in severe chorea. These two facts and the general character of the movements suggest that although the resemblance to chorea is striking it is mainly superficial and the presence of the cranial nerve palsies confirms this view. The tendon jerks are all sluggish, but obtainable. They are equal on the two sides. Abdominal reflexes doubtful. Plantars flexor. There is no demonstrable defect of sensation. She complains of pain in the region of the right wrist, but no local abnormality of any kind can be detected.

(June 2, 1920.) After eighteen hours the movements ceased suddenly and the patient subsided into a profound lethargy. She now resembles the ordinary form in which lethargy and signs of a peri-aqueductal lesion co-exist. Temperature remains raised. She can be aroused for feeding and to answer questions. The pain in the right wrist has gone.

June 15, 1920. During the past two weeks the cranial nerves have rapidly regained normal function. There is now very slight ptosis. The eye movements are normal and there is no facial weakness. She is still somewhat lethargic and sleeps most of the day. At night she is more wakeful and complains of pain in the region of the left shoulder. This pain is localized in distribution and not neuralgic in character. There is no local condition to account for it. The rash has disappeared and the general condition is good.

On admission the cerebrospinal fluid was examined. There were 160 lymphocytes to the cubic millimetre. There was no leucocytosis in the blood.

The extreme restlessness accompanying the delirium when the patient first came under observation closely resembled severe chorea, and it was with this diagnosis that she was admitted to hospital. The presence of cranial nerve palsies, the sudden onset, and certain minor features which are emphasized in the foregoing account sufficed for the recognition of lethargic encephalitis.

The extreme violence of the movements, their incessant and disorderly character and their association with marked psychical excitement make it extremely probable that we are dealing here with an irritative lesion of higher cerebral centres, rather than any release mechanism.

Case 4.—*Lethargy and Acute Cerebellar Ataxy.*

General symptoms.—None observed.

Nervous symptoms.—(General) Acute onset of headache, vertigo and vomiting. (Focal) Acute cerebellar ataxy.

Captain F. C., aged 27. On May 5, 1920, after a hearty breakfast, and having been previously in apparently excellent health, he was suddenly seized with severe headache, giddiness and vomiting. These symptoms persisted throughout the day. He was examined during that evening and then lay curled up on his bed on his right side. He showed extreme photophobia, but was not irritable. He was fully conscious and rational, but could not give a good account of his symptoms on account of a gross defect of articulation. His friends stated that he had "stammered badly" all that day, though he had never been known to do so before. There was no fever, pulse rate was 64, and respiration rate 20.

Re-examined in hospital on May 7, 1920. His general condition was improved, the vomiting had ceased and the headache diminished, though some occipital pain remained and was aggravated by movement. There was no sense of giddiness. He was distinctly lethargic and lay curled up in bed and apparently asleep until spoken to, when he became quite alert and rational.

There was extreme ataxy of articulation, which was slow and accompanied by overaction of the facial muscles.

Vision: acuity, fields and fundi, normal. Hearing normal, some tinnitus. The pupils were equal and reacted normally. There was no ptosis, or diplopia, but there was a slight divergent squint of the right eye, probably congenital. On lateral deviation a fine rapid nystagmus appeared.

There was a doubtful paresis of the lower part of the left side of the face. Deglutition and phonation were normal. The tongue was protruded normally.

There was gross ataxy of the arms of cerebellar type, the right arm was particularly unsteady and jerky on movement. He walked with a wide base and had to be supported. There was no tendency to fall in any constant direction. The tendon reflexes were brisk and equal on both sides, the

abdominal reflexes were present. The left plantar reflex was doubtfully extensor in type. There was no discoverable defect of sensation.

The cerebrospinal fluid was normal in cell and globulin content. The Wassermann reaction was negative. There was no leucocytosis in the blood.

During the six weeks after admission all these symptoms gradually cleared up and on discharge in June slight ataxy of speech, which was still somewhat staccato in character, and some intention tremor of the right hand and arm, were the only noticeable symptoms. When fatigued some unsteadiness of gait appeared.

In this case the focal symptoms are those known to be associated with a negative or defective lesion of the cerebellum. They are in no way peculiar to lethargic encephalitis.

Case 5.—Lethargy and Signs of Irritation of Lower Motor Neurones.

General symptoms.—Lassitude, anorexia and constipation.

Nervous symptoms.—(General) Insomnia, nocturnal delirium, hallucinations passing into somnolence. General muscular hypotonus and diminished tendon jerks. (Focal) Rhythmic contractions of certain arm and abdominal muscles.

George C., aged 44. (History obtained from friends.) Had a "chill" last April. Since this he has not been well. He has complained of being tired, without appetite, and constipated. On May 26 he began to be sleepless at night and to mutter in his sleep. During the following week he complained of severe pains in his limbs, was told by his doctor that he had rheumatism and advised to stop work. Early in June he had severe pain in the chest and was thought to have pleurisy. Since the first week in June he has been confined to bed, semi-unconscious during the day and restless and violent at night. He appeared to be having terrifying visions, talked excitedly and had to be kept in bed by force. He was incontinent of urine and very constipated.

On admission to hospital (June 19, 1920) he was profoundly lethargic, but could just be roused to answer simple questions. He could give no account of his illness. He understood spoken speech perfectly and replied in whispers. He complained of no pain. He passed urine in the bed. Temperature was 101° F., pulse 128, and respirations 32.

Hearing and vision appeared normal. Fundi were normal. The cranial nerves showed no defect. There was general muscular weakness and hypotonus, but no localized paresis. There was no spontaneous tremor or muscular contraction. No sensory loss could be detected. The tendon jerks were all very sluggish, the abdominal reflexes not obtainable and the plantars flexor.

The cerebrospinal fluid was normal in every respect, and there was no leucocytosis in the blood.

During the following two months his general condition and his lethargy remained unchanged and he became slowly thinner. It was found during this period that on passive manipulation of the arms, and after such forceful voluntary contractions of the muscles of these limbs as he could be induced to

carry out, there appeared slow clonic contractions of certain forearm and small hand muscles. The rate of these was fairly constant and was about 48 per minute. These contractions which involved the whole muscle appeared first in the supinator longus, then in the extensor group of muscles, and finally in the thenar muscles. They were feeble, and after two or three minutes they died away until the limb was further manipulated.

Similarly, abdominal palpation evoked feeble rhythmic contractions of the upper segments of the recti on both sides, which caused the umbilicus to be drawn upwards at each twitch. Very occasionally the right sternomastoid was seen to twitch spontaneously in the same manner and at the same rate.

September 1, 1920. The patient's general condition is gradually failing, he is losing weight and a large sacral bedsore has appeared. The lethargy remains unchanged and he can still be aroused and can carry out simple orders and answer simple questions in a whisper.

The rhythmic contractions described above have undergone an interesting change. Passive stretching of the forearm and hand muscles now evokes fibrillation and occasionally fascicular twitching of large strands of muscle. If passive manipulations be maintained for several seconds a slow rhythmic contraction, very feeble and producing very little movement of the digits, of the flexor muscles in the forearm appears and lasts for some seconds. Neither fibrillation nor contraction can be induced in the upper arm or the lower limb muscles, nor in the recti abdominis.

The only somatic nervous symptoms observed in this case were fibrillary and fascicular twitching and rhythmic contractions of certain muscles. The distribution of the affected muscles was quite random, and there was no evidence of synergically associated muscles being affected. Moreover, the contractions were for the most part not spontaneous, but induced by passive manipulation of the muscles, and it seemed that as the tendency to "myoclonus" abated fibrillary and fascicular twitching took its place.

The random distribution of the clonic contractions in muscles not functionally associated, its restriction in certain circumstances to segments, strands, or even smaller muscle-fibre bundles (fascicular and fibrillary twitching), indicate very definitely that we are in all probability not dealing with a lesion of neurones of high physiological levels, but with an affection of the lower motor neurones, presumably irritative in character.

Case 6.—*Delirium, Confusional State, Restlessness and Pain followed by Signs of Irritation of Lower Motor Neurones.*

General symptoms.—"Influenza" followed by persistence of pains in back and limbs, fever, swelling of ankles, albuminuria and constipation.

Nervous symptoms.—(General) Headache, insomnia, restlessness, delirium

and confusional state, general muscular weakness and hypotonus. (Focal) Rhythmic contractions of isolated muscles.

Gertrude B., married, aged 38. In March, 1920, patient had an illness, of which no details are available, which was diagnosed as influenza. Following this she did not recover completely, but complained of lassitude, fleeting limb and back pains, some fever at night (100°), swelling of the ankles and sleeplessness.

When seen on April 5, 1920, nothing abnormal was noted beyond œdema of the ankles and marked albuminuria.

On April 10, 1920, she became "light-headed," and complained of severe pain in the right arm, epigastrium and back.

On April 12, 1920, an erythematous rash appeared on abdomen, back and arms, with forcible twitching of muscles of neck, upper limbs and shoulders. This was accompanied by severe pain in the affected muscles. The temperature was 100° F.

On April 14, 1920, she was less restless and excited, but was very confused as to her whereabouts and did not recognize her relatives.

On April 17, 1920, the albuminuria had disappeared, the temperature remained slightly raised. The rhythmic muscular contractions persisted and she was restless and delirious at night.

She was admitted to hospital on May 31, 1920. She was pale and generally wasted and had a harassed, anxious expression. There was no trace of lethargy. She was quite disoriented as to time and place, and identified those in charge of her with various members of her family. She was inclined to be talkative and gave extravagant accounts of her doings during the day. Her mental state was like that seen in Korsakow's psychosis. Speech was normal. Hearing and vision were normal.

The pupil and their reactions were normal, the ocular movements were normal. There was no ptosis, squint, diplopia or nystagmus. All the cranial nerves were normal,

There was general wasting, weakness and flaccidity of the muscles, but no localized paresis. All movements were unsteady, probably on account of the extreme general weakness. There was no tremor. The weakness was so marked that she was very helpless and could do nothing for herself. No sensory change could be detected. The tendon jerks were all brisk, the abdominal reflexes were present and equal on the two sides. The plantar reflexes were doubtfully extensor on both sides. There was no sphincter defect.

Examination of the abdominal wall revealed a constant rhythmic contraction of the lower segments of the recti abdominis, particularly on the right side. This caused a rhythmic downward movement of the umbilicus (downwards and to right). At times, usually while she was asleep, the left sternomastoid showed a similar clonic contraction. The rate of the beats in both instances was 48 per minute. The abdominal muscle twitching undoubtedly increased in force when the muscles were palpated, and even when the patient was being

examined and was consciously under observation, but it continued during sleep. It was noteworthy that if the abdominal reflexes were rhythmically stimulated at a faster rate than that of the spontaneous twitching, the rhythmic reflex responses completely replaced the spontaneous beats for the time being; that is, both movements did not occur simultaneously, one or the other monopolized the muscle.

The cerebrospinal fluid at the time of admission was clear and free from excess of cells (average of two cells per c.mm. in several counts).

The patient remained under observation for two months. During this time her mental state gradually became normal. There was at no time either lethargy or cranial nerve palsies. Her general condition steadily improved and she regained strength and weight.

The abdominal muscular twitchings became less forceful, but persisted throughout and were still present when she left hospital at the end of July.

In this case, as in Case 5, the sole somatic nervous symptoms indicated an irritative lesion of lower motor neurones. The considerations advanced in the previous instance apply here and indicate that we are dealing with involuntary muscular contractions differing in nature and site of origin from the clonic convulsive movements seen in Case 1.

Case 7.—Signs of Irritation of Sensory and Motor Spinal Roots.

General symptoms.—None observed or recorded in history.

Nervous symptoms.—(General) None observed or recorded in history. (Focal) Intense neuralgic pain in distribution of cervical sensory roots, followed by the appearance of persistent fibrillary twitching in muscles of right shoulder girdle and arm.

Charlotte M., married, aged 36. On about June 20, 1920, having gone to bed feeling perfectly well, she awoke at 2 a.m. with intense pain "in the spine of the neck" running up to the back of the head. The muscles at the back of the neck "seemed to be all on the work" and her head was drawn backwards. So severe was the pain that she had to cry out aloud.

In the morning the pain had spread to the right shoulder and seemed settled on the right side of the neck also. It was still intense and seemed to shoot outwards from the back of the neck down on to the shoulder and the upper part of the right side of the chest. She noticed that all the muscles round the right shoulder, under the right breast and in the arm as far as the elbow "were all on the twitch." Her friends "could see them wriggling through her blouse." These twitchings did not move the limb at all, but the muscles looked "as though there were worms wriggling beneath the skin." During the day the "wriggling" spread to the muscles of the forearm, and the whole limb became weak, so that she could not lift her arm up to her mouth, and her grasp became unsteady and weak. The limb felt very heavy. This

condition persisted for two days and then became very much less marked, though even yet when she uses the arm they start again round the shoulder. The right arm has remained weak, she cannot hold a pen or a needle partly on this account and partly because the hand is unsteady and shaky.

The neuralgic pains persisted unabated for two weeks. During this time she could not rest for them, she slept hardly at all, and had to walk about constantly with the right arm supported to ease the pain. For the past three weeks the pain has gradually diminished, though even yet she cannot lie on the right shoulder without bringing it and the twitchings on again.

There was no lethargy at any time. There is no history of cranial nerve palsies, nor of any constitutional disturbances, such as fever, headache, constipation, nausea or vomiting.

She states that she has always been highly strung. This has been much worse since the air raids, which have made her very nervous and easily upset. Her general health has always been good. She has had three pregnancies and three healthy children born at full term. Two died during the first year of life from "convulsions," and the third is alive and well.

On admission to hospital (July 26, 1920) she was found to be a well-nourished woman of healthy appearance. Thoracic and abdominal viscera healthy. Intelligence and emotional tone normal. Vision and hearing were normal. There was no defect of cranial nerves.

The motor system: There was weakness and extreme tenderness to pressure of the following muscles of the right shoulder girdle and arm: sternomastoid, trapezius, all the scapular muscles, pectoralis major, deltoid, the rhomboids, triceps, biceps. The forearm and small hand muscles were slightly tender. There was some tenderness of serratus magnus and distinct winging of the scapula. There was general hypotonus of the affected muscles. All movements of the limb were limited in range on account of weakness, and active movements produced a tremulousness of the hand and arm that was partly due to this weakness.

Active or even passive movements of the various segments of the limb produced at once ample fascicular and fibrillary twitching of the muscles involved, and the contractions partook of the characters of both fibrillation and clonus. For although the twitchings were regular in frequency, they involved varying proportions of a muscle in different contractions, so that contractions of small muscle bundles, of strands of muscle running the whole length of the muscle and of the whole muscle simultaneously were seen. These contractions did not cause actual movement of segments of the limb.

There was no other abnormality of the musculature elsewhere in limbs or trunk.

Over the upper arm and fore-quarter on the right side there was a qualitative alteration of cutaneous sensation. All touches were felt, and pinprick was painful, but sensations appeared diffuse and not accurately localized and differed in quality from normal areas of skin. This area could not be accurately mapped out.

The tendon jerks in both arms were present and no definite inequality between the two sides could be made out. The knee and ankle jerks were normal, the abdominal reflexes present and the plantars flexor in type.

The patient was only under observation on two occasions during the last week in July and her present condition is not known.

This case did not come under observation till convalescence was well established, but nevertheless the physical signs were in accord with the history of the early stages of the illness. They were those of an irritative lesion of cervical spinal roots, both motor and sensory. If this view of the localization and character of the lesion be correct, it confirms the hypothesis that the "myoclonic" and fibrillary twitchings recorded in the two previous cases are dependent upon irritation of the lower motor neurones. In these the absence of definite tenderness of the twitching muscles and of neuralgic root pains suggests that the lesion was in grey matter, rather than in the roots.

Sicard [20] records an exactly similar case.

III.—Involuntary Movements and Muscular Contractions; the so-called "Myoclonic" and "Choreiform" Manifestations of Lethargic Encephalitis considered as Irritative Phenomena. The Contrast between the Paralytic and the Exciting Action of the Virus.

The general impression derived from the study of this small series of cases accords with that obtained from a review of the literature of lethargic encephalitis: namely, that the constitution of the symptom-complex in any given case appears to be largely fortuitous and to follow no laws that we can yet determine. Any and every part of the nervous system may be involved and there is no combination of symptoms that we may not encounter. In consequence, many cases cannot be placed in any type unless types are to be multiplied until they cease to have either value or significance. It will be suggested later, however, that certain general principles do govern the clinical manifestations of this disease.

It is not now proposed to dwell in detail upon each of the many points for discussion arising out of these cases, and the particular aspect of the disease to which some consideration will be devoted in this paper is that introduced by the appearance of the motor symptoms mentioned in the heading to this chapter.

However, before any attempt is made to draw conclusions as to

their origin and nature in the particular cases recorded here, it may be of interest to turn to the numerous published descriptions of similar cases for any useful indications they may afford in this respect. The result is somewhat disappointing, for although many comprehensive accounts are to be found, particularly in the French literature, the clinical details given are so frequently coloured by the effort to identify widely differing muscular manifestations with the "secousses myocloniques" of Sicard and Kudelski [17, 18] that their probable nature can often only be surmised.

A case reported by Roger and Aymés (16) will help to illustrate this. It is described as a "syndrôme hémimyoclonique alterne séquelle d'encéphalite épidémique."

A young adult of 22 had an acute illness with convulsions followed by the development of a right-sided hemiparesis (face, arm, leg), with inco-ordination of the right upper limb, clonic jerkings of the right arm and slightly of the right leg. The right tendon jerks were increased and the right foot showed an "intermittent" extensor response. There is said to have been spasm of the left side of the face.

It is highly probable that we are dealing here with an encephalitic lesion of the left cerebral motor cortex with Jacksonian attacks in the right limbs. Again, we find Souques [21] expressing the view that "entre l'encéphalite myoclonique et l'encéphalite choréique, il n'y a, en effet, que des différences de mesure et de rythme dans les mouvements involontaires." Achard [1] has expressed a similar view. While, finally, Comby [8] describes as myoclonic encephalitis an illness with onset of fever, malaise, torpor followed by a phase of "myoclonus" and restlessness. The involuntary movements are described as being incessant, irregular and disorderly. Here we find the convulsive movements of what are almost certainly a Jacksonian fit described as "myoclonus," the view expressed that there is no real distinction between myoclonic contractions and choreiform movements, and in Comby's paper we see the latter described under the same name as the former.

From all this it is clear that the terms "myoclonus" and "myoclonic" in connection with this disease, though precise enough in Sicard's hands, have in those of others ceased to have a specific meaning and frequently serve only to obscure the characters of the involuntary contractions and movements described in clinical reports of this disease.

In these circumstances it may be of value to consider the original

meaning of the term myoclonus. It was first employed by Friedreich in 1881 to describe certain features seen in the disease he named paramyoclonus multiplex. "This affection," states Oppenheim, "is characterized by clonic contractions affecting mainly the muscles of the extremities and trunk, and rarely if ever those of the face. The contractions are short and lightning-like; they involve a small number of muscles which do not have a synergic action. The effect of the contractions in moving the limb is therefore slight or entirely absent. The contractions involve the muscles of the two sides of the body almost equally; they may be symmetrical, but not synchronous or rhythmical. The various twitchings, of which there may be sixty to one hundred a minute, are separated by intervals of varying duration. They affect a single muscle, which cannot be voluntarily contracted by itself, e.g., supinator longus. They may even be limited to parts of a muscle . . . active movements, which as a rule are unaffected, have a tranquillizing, soothing effect upon the spasm. Emotion has the opposite effect. The twitching diminishes when the attention is distracted, and disappears entirely during sleep." It may be added in conclusion that no discoverable lesion of the nervous system underlies the phenomena thus named and described.

In his first paper on the subject [17], Sicard describes the contractions in his myoclonic encephalitis as "secousses musculaires, brèves, rapides, explosives, à type rythme électrique, qui siègent sur la musculature des membres, de la face et du diaphragme, tantôt myocloniques, frappant un muscle ou un groupe de muscles, et ne s'accompagnant pas de contractions fibrillaires. Les algies disséminées persistent."

A comparison of Oppenheim's account with that of Sicard reveals the fact that the muscular contractions in the two cases bear but a relative resemblance, show certain distinct points of difference, and as far as our present knowledge goes appear to differ completely in cause.

Even, therefore, if we allow its aptness, the term myoclonus has but a limited sphere of usefulness in the study of lethargic encephalitis, and unless it is employed in Sicard's sense alone, and as a purely descriptive term without pathological basis, it had almost better be abandoned.

Nevertheless, from the descriptions given by those observers who use the term precisely we may obtain some useful information as to the probable site of the origin of these clonic contractions. Perhaps the most direct indication is to be found in two cases reported by

Sicard [17, 19], in which clonic contractions in the upper limbs gave place after several days' persistence to a paresis of peripheral type, namely, extensor weakness with wrist-drop. In one of these it is recorded that supinator longus was spared. While Sicard did not observe fibrillation associated with these contractions, such a combination has been recorded by several observers (Carnot et Gardin [6]). Again, we may note the random distribution of the clonic contractions, their frequent association with neuralgic pains of peripheral and spinal root distribution (often coinciding with that of the muscular contractions in topography). All grades of rhythmic muscular excitation have been recorded; fibrillary twitching, fascicular twitching (paramyoclonus) and contractions of whole muscles and muscle groups. These features all point to the lower motor neurone as the seat of an irritative or exciting lesion. According to Netter, "myoclonic encephalitis" marks the appearance of irritative symptoms in a disease where previously paralytic symptoms have predominated. However, neurologists have become cautious of explaining involuntary movements, and in some instances even pains, by postulating irritative lesions. Indeed, we know that the pain of the thalamic syndrome and the involuntary movements of tremor and athetosis are probably not so caused, but depend upon the release of lower centres from control. They are a reaction, an abnormal over-activity of intact portions of the nervous system when higher controlling mechanisms are out of action from destruction or otherwise. In the present instance it is extremely unlikely that we are dealing with any such release mechanism, and for the reasons given above it seems more probable that in the case of these clonic fibrillary, fascicular and muscular contractions we see the expression of an irritation or excitation of the lower motor neurone. The occurrence of Jacksonian fits indicates that the virus of lethargic encephalitis can and does act in this way upon nerve cells.

In considering the seven cases recorded in this paper, it is not proposed to discuss all the motor symptoms described, such as the Jacksonian fits of Case 1, the tremor and rigidity of Case 2, or the cerebellar symptoms of Case 4. These are in no way peculiar to lethargic encephalitis, nor does their occurrence in this disease throw any fresh light on their nature or origin. It may be of interest, however, to discuss briefly the choreiform movements of Case 3 and the fibrillary and muscular twitchings seen in Cases 5, 6, and 7.

In the preceding paragraphs and in the commentaries on the three last named cases reasons have been given for regarding the so-called

myoclonic contractions and the fibrillary and fascicular twitchings with which they are frequently associated, as evidence of an irritative or exciting action of the virus of the disease upon the lower motor neurone; that is, upon the cells of the peripheral motor nerves. It is now further suggested that the extreme psychomotor excitement of certain cases of lethargic encephalitis is another example of this action of the virus, in this case upon the highest cerebral centres. If this view be correct we see in the psychical and in the choreiform motor manifestations, in the Jacksonian fits, and in the rhythmic contractions and fibrillary twitchings of various muscles and parts of muscles the expressions of irritation or abnormal excitation of neurones in each of Jackson's three physiological levels of the nervous system.

There are many reasons for regarding the choreiform agitation (the "folie musculaire" or the "syndrome choréique à grand fracas" in the expressive language of the French observers) of such a case as the third of the present series as being irritative rather than dependent upon a release mechanism. Its constant association with great psychical excitement, its frequent association with the clonic muscular and fibrillary contractions already discussed (Labbé et Hutinel [13], Sicard [20], Harvier et Levaditi [11], Ardin-Delteil et Raynaud [2], Kahn [12], Dupouy [9], and Bourges et Marcandier [4]), and its rapidly fatal termination in most instances in exhaustion, coma and death all point to its being of this nature. While the disorderly, varying and complex character of the movements not only indicates that they arise in centres of high physiological level, but also marks them off clearly from the more orderly involuntary movements of the tremor and athetosis varieties, which are accepted as depending upon removal of control over subordinate motor mechanisms.

Finally, if we can accept this view of the irritative origin of these motor phenomena another remarkable point claims attention. It is that the virus of lethargic encephalitis in its paralytic action on the nervous system has always shown a characteristic and marked selective affinity for certain groups of motor neurones in the basal ganglia and brain-stem, producing the familiar basal ganglia and midbrain types of the disease with their associated lethargy. It does not cause widespread random paralyses, such as we meet in acute poliomyelitis. In its exciting or irritating effect, on the other hand, this selective affinity is totally absent, and it seems that neurones of every level physiologically considered, or, speaking anatomically, every part of the nervous system from cerebral hemispheres to spinal roots, are indifferently attacked.

Thus the polymorphic clinical characters of lethargic encephalitis are in large measure due to this second action of the virus, and we have in effect irritative and paralytic forms or stages of the disease. Each possesses clinical features of its own. Both may be present in a given case, and they occur in numerous and widely varying combinations, or they may occur singly. However, even in purely paralytic or negative forms of the disease, the involvement of the nervous system is probably more widespread than the focal symptoms indicate. The frequent general muscular hypotonus and diminution of tendon jerks of the lethargic paralytic cases would suggest this view.

Nevertheless, the striking contrast between these two effects of the virus on the nervous system raises a question which it must remain for pathological investigation to answer; namely, whether we may not be dealing with a complex and varying virus containing more than a single active component.

IV.—Summary and Conclusions.

The protean manifestations and the remarkable clinical diversity of lethargic encephalitis are discussed, and it is suggested that the numerous schemes of classification and description adopted, though inevitable in the early and growing stages of knowledge of so polymorphic a disease, are in fact tending to confuse rather than to lend precision to our conceptions of this malady, for practically every case presents, either simultaneously or at some phase of its course, the features of several clinical "types." The example of "myoclonic encephalitis" is discussed as demonstrating the misleading results of a purely symptomatic basis of classification and description.

A natural grouping of the symptoms of any toxic infective disease of the nervous system is given, and it is suggested that description, and as far as possible classification, upon an anatomical basis—that is, using the focal nervous signs for the purpose of defining types—would obviate much of the confusion at present to be found in the literature, and would at least ensure a uniform terminology in descriptions of the disease.

Acute poliomyelitis and lethargic encephalitis are contrasted in the light of the scheme referred to.

The points thus discussed in the first chapter are illustrated by a small series of personally observed cases, and an account of various positive motor symptoms is given. It seems probable that the psychomotor excitement of the "choreiform" manifestations of lethargic

encephalitis, the Jacksonian fits, and the rhythmic fibrillary, fascicular and muscular contractions collectively grouped by various writers as "myoclonic" symptoms are in fact the expressions of an irritative or exciting action of the virus of the disease upon neurones of the three physiological levels (Hughlings Jackson) of the nervous system, and that such symptoms can be localized on the same principles as are employed to localize negative or paralytic symptoms. If we accept this view of the irritative origin of the motor symptoms under discussion, a striking feature emerges: namely, that where the virus produces negative or paralytic symptoms it has, ever since the first appearance of the disease, shown a definitive and characteristic selective action on the cells of the basal ganglia and on those of certain motor nerves in the brain-stem, producing the familiar basal ganglia and midbrain types of the disease with its associated lethargy. On the other hand, in its irritative or exciting effect the virus appears to act equally on any and every part of the nervous system, from cerebral hemispheres to spinal roots, hence the polymorphic character of cases showing what we may call irritative symptoms. It is such cases that present the many difficulties in any attempt to classify lethargic encephalitis into clinical types. Possibly we may be dealing with a complex virus in which more than one active component exists.

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ON DECEREBRATE RIGIDITY IN MAN AND THE OCCURRENCE OF TONIC FITS.

BY S. A. KINNIER WILSON.

FOR a number of years certain analogies between the results of experimental mesencephalic transection in apes and other mammals and occasional clinical phenomena in man, in cases where cortical function is completely in abeyance, have appeared to me worthy of consideration. Such cases, usually of an acute nature, do not occur with any frequency, and since 1905 only some half dozen of this particular character have presented themselves, but their main features exhibit similarities so definite as at once to suggest a more or less identical origin and localization of the symptoms. Briefly, the type of case herein described and analysed is one in which there is evidence of withdrawal of cortical control in the form of unconsciousness or semi-consciousness, the result commonly of cerebral hæmorrhage—submeningeal or intra-ventricular, or both—or of meningeal inflammation, or from the effect of certain intracranial tumours; also, for that matter, of hysteria. Coupled with this impairment of consciousness has been the appearance of tonic rigidity of trunk and limbs, minutely resembling experimental decerebrate rigidity, to which frequent exacerbations in the form of tonic or postural fits, accentuating and exaggerating the background of tonic posture, are superadded.

It will be shown, further, that in certain types of organic and functional nervous disease these postural attitudes may be adopted involuntarily with full consciousness, and that partial or fragmentary types also occur. To this last statement much importance is attached, since the hypothesis to be subsequently developed is, that the involuntary postures of a limb or segment of a limb in many forms of nervous disease, as well as the transient positions assumed in involuntary movements such as chorea or athetosis, are in reality simply parts of the complete decerebrate attitude.

The material may be divided into two sections as follows :—

I.—(1) Cases of decerebrate rigidity or attitude and tonic fits combined.

(2) Cases of decerebrate attitude without tonic fits.

(3) Cases of tonic fits without persisting decerebrate attitude.

These three groups include only those cases where the phenomena appear in association with loss of consciousness; their significance is enhanced by the light they throw on the second section of the material:—

II.—(1) Cases of decerebrate attitude in conscious life.
(2) Incomplete, limited, or fragmentary cases.
(3) Cases of decerebrate attitude in the course of conscious involuntary movement, especially chorea and athetosis.

I wish here to express my indebtedness to various colleagues who have kindly allowed me to examine or to refer to cases which have been under their care, and to acknowledge at the outset the debt this paper owes to the genius of Dr. Hughlings Jackson and of Professor Sherrington. It has proved a matter of considerable difficulty to collate from the literature analogous cases to those herein described, mainly, no doubt, because of the usually quite incidental reference to the phenomena and the variety of the headings under which such cases might be expected to be found. Those of which a *précis* is given later provide valuable confirmation of the general views expressed in this article.

To appreciate the significance of the clinico-pathological cases more readily it is desirable to cite first the results of experimentation.

The salient features of experimental decerebrate rigidity, as contrasted with the state following on transection below or in the lower half of the medulla, are given by Sherrington [15] as follows, the latter condition being described first:—

"If in a monkey or cat transection below or in the lower half of the bulb has been performed, the animal . . . . hangs from the suspension points with deeply drooped neck, deeply drooped tail, and its pendent limbs flaccid and slightly flexed . . . . On giving the hand or foot a push forward and then releasing it the limb swings back into and somewhat beyond the position of its equilibrium under gravity; and it oscillates a few times backward and forward before finally settling down to its original position.

"To this condition of flaccid paralysis supervening upon transection in the lower half of the bulb the condition ensuing on removal of the cerebral hemispheres (mesencephalic transection) offers a great contrast. In the latter case, the animal, on being suspended just in the same manner as after the former operation, hangs with its fore-limbs thrust backward, with retraction at shoulder-joint, straightened elbow, and some flexion at wrist. The hand of the monkey is turned with its palmar face somewhat inward. The hind-limbs are similarly kept

straightened and thrust backward; the hip is extended, the knee very stiffly extended, and the ankle somewhat extended. The tail, in spite of its own weight . . . is kept either straight and horizontal or often stiffly curved upward. There is a little opisthotonos of the lumbo-sacral vertebral region. The head is kept lifted up against gravity, and the chin is tilted upward under the retraction and backward rotation of the skull. . . . . When the limbs or tail are pushed from the pose they have assumed, considerable resistance to the movement is felt, and, unlike the condition after bulbar section, on being released they spring back at once to their former position and remain there for a time even more stiffly than before."

Attention is specially directed to the posture of the fore-limbs in extension-pronation, and to the head-retraction and opisthotonos.

We may now pass to an examination of cases observed in man.

I.—(1) Cases of Decerebrate Rigidity or Attitude and Tonic Fits combined.

Case 1.—W. S., aged 19, shopman, was admitted to the National Hospital on June 19, 1905, under the care of Dr. J. A. Ormerod.

For at least four weeks previous to admission he had suffered from severe headaches, mainly frontal. About 11 p.m. in the evening of June 11, 1905, he suddenly "felt his head bad" and fell in the street unconscious, remaining so for about twenty minutes; thereafter, he complained of splitting headache across the forehead. During the night he had a fit with convulsions, but details were not forthcoming; it was followed during the next twelve hours or so by about nine other fits, apparently of a general epileptiform nature. In the course of the week before admission he frequently vomited and complained of giddiness, and was twice incontinent of urine. On getting up and attempting to walk he was stated to have staggered badly.

On admission to hospital his general condition was as follows: He lay on his back in bed, frequently passing his right hand over his forehead in a half-unconscious fashion. All questions were answered rationally, but usually in monosyllables, without dysarthria. He was drowsy and indifferent to his surroundings: frequently he gave long-drawn sighing expirations: frequently, also, he would snort and spit. While he often moved the right limbs about, the left were immobile; the left arm was kept adducted and flexed at the elbow, and the left leg adducted slightly at hip and flexed slightly at knee.

Well-marked double optic neuritis was present. The pupils reacted quickly to light. There was apparently some general weakness of all ocular movements, but the patient's state did not admit of satisfactory testing. In voluntary movements the right side of the face always moved before the left. The tongue was protruded in the mid-line.

In addition to the apparent paralysis of the left limbs, it was found that the left arm showed distinct rigidity and resistance to passive movements, and the left leg similarly. No sensory changes were detected. The arm jerks were equally diminished on the two sides. The abdominal reflexes were much down on the left. Knee and Achilles jerks were diminished on both sides, and a well-marked double extensor response was readily obtained, but no ankle clonus could be elicited. Occasional incontinence of urine was noted.

By lumbar puncture 20 c.c. of fluid were drawn off under considerable tension, pale, reddish-yellow in colour, quite translucent, and obviously hæmorrhagic. There was no difference between the first drop and the last.

The next day the patient was able, after much encouragement, to move the left arm a little at shoulder and elbow, and to make a feeble grasp with the left hand. On the other hand, the facial asymmetry on volitional movement was accentuated. Ocular movements were still imperfect; the sixth nerves were apparently weaker than the others, slight internal strabismus being noted with the eyes at rest. The knee-jerks were further diminished, especially the left, and the double extensor response persisted.

A diagnosis of cerebral tumour was made, probably right frontal, and, further, it was presumed that for some reason slight hæmorrhage into and from the tumour had taken place when the symptoms had become acute just prior to admission.

At this point, however, they changed, and presented the following striking clinical picture.

On the same day, June 23, 1905, at 11.10 p.m., I was called to see the patient.

He was lying on his back, with head turned slightly to the right, and was quite unconscious. The arms were extended by the sides, slightly flexed at the wrists, and notably rigid. The legs were also extended and adducted, with toes pointed down and in. Respiration was extraordinarily laboured, the rate no more than five to the minute; inspiration was short, about three seconds' duration, while expiration was long drawn out, averaging nine seconds. It was noisy at first, gradually becoming fainter and fainter, then a sudden jerky inspiration completed the cycle. The pulse was 180, full, of moderate tension, and slightly irregular, sometimes thudding for several quick beats, then intermitting. With inspiration it always beat more quickly and strong.

The pupils were regular and equal, about 4 mm., and reacted to light. The corneal reflexes were absent. When the lids were lifted the eyes were seen to be fixed and staring straight in front. Skin and mucous membranes were flushed but not cyanotic.

11.15 p.m. Respiration remained about 6; pulse had fallen to 96, and was again regular. The knee-jerks could not be elicited on either side, but a smart patellar tap produced in each case a contralateral adductor jerk. The legs remained fully extended and rigid. The recti abdominis were also rigid Patient was incontinent of urine, and a minute later had an erection.

11.20 p.m. The eyes began to turn slightly downwards, and at the same time the right pupil dilated till it was almost twice the diameter of the left; it was now immobile to a bright light, though the left contracted as before. The plantar responses, up to this point absent, were now extensor on both sides.

11.30 p.m. The eyes gradually deviated into a definite and unmistakable skew-position, the right eye being down and in, the left eye up and out. Both pupils reacted again to light, though the right remained larger than the left. The skew-deviation was maintained only for a minute and a half, both eyes then slowly moving to a more symmetrical position, while the right pupil became of the same size as the other.

11.45 p.m. About this time both arms became rigid, the left rather more than the right. Both were adducted at the shoulder and strongly extended at the elbow, with pronounced internal rotation and hyperpronation; the wrists were flexed, so that the palms of the hands looked up and out, quite turned

FIG. 1 (Case 1).—The typical decerebrate attitude of extension-pronation.

away from the body (fig. 1). This striking position of the arms was henceforward maintained; at intervals the whole arm would stiffen still further, as it were, and the attitude become more accentuated, as if by waves of contraction passing down the musculature, but even when these waves relaxed the extensor-pronation position was not abandoned. The legs were as before, rigid, adducted, extended, feet inverted and great toes strongly dorsiflexed.

12.20 a.m. Another fit started. Patient suddenly became absolutely stiff in the decerebrate position, and his face reddened, while respiration became occasionally laboured, down to four per minute. It consisted of a short inspiration followed by a long-drawn-out expiration in the proportion of about 1 : 4. The pulse had become much more rapid again, 140 in rate, but it was fairly regular. There was no twitching of the limbs, but simply an intense tonic spasm accentuating the extensor attitude. The eyes were open, staring straight in front; pupils were dilated to their widest and did not react to light; corneal reflexes were absent on both sides.

FIG. 2 (Case 1).—The typical attitude at the summit of one of the actual tonic fits. The left forearm is less pronated than the right. Intense tonic contraction of the extensor musculature, with slight wrist flexion.

[For figs. 1 and 2 I am indebted to Dr. Colin Russel, of Montreal, who was in residence with me at Queen Square at the time.]

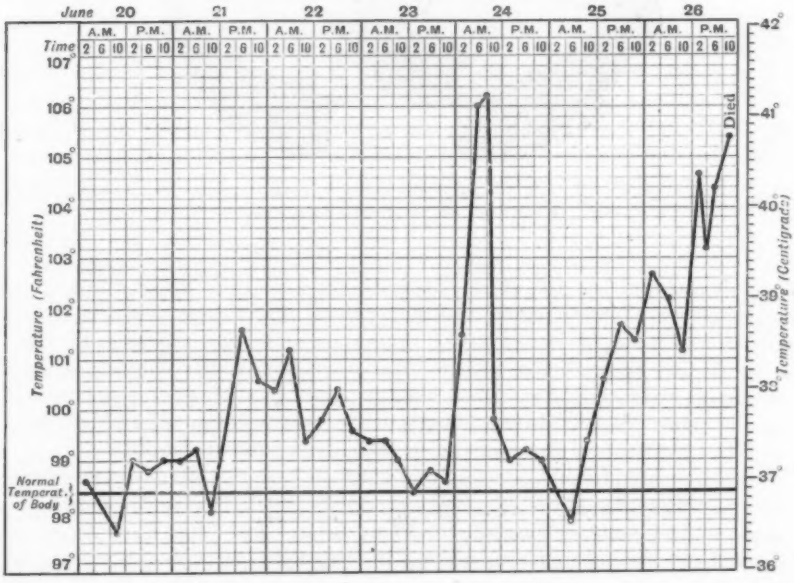
June 20 21 22 23 24 25 26
A.M. P.M.
Time 2 6 10 2 6 10
107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°
Normal Temperat. of Body
Temperature (Fahrenheit)
Died
42° 41° 40° 39° 38° 37° 36°
Temperature (Centigrade)

FIG. 3.

1.25 a.m. When the patient was seen again, after about an hour's interval, he was in the same extensor position, arms adducted at shoulders, extended at elbow, flexed at wrist, whole arm rotated inwards and hyperpronated; on the right the thumb was between the first and second fingers of the hand, but not on the left (fig. 2). The legs were rigidly extended, feet inverted, great toes dorsiflexed. Respiration 9, pulse 130.

For the remaining two days of the patient's life the phenomena continued at very frequent intervals without cessation. It appeared that any handling of the patient, e.g., during sponging (for with the decerebration high irregular temperature occurred, see fig. 3), precipitated the "attack," which consisted,

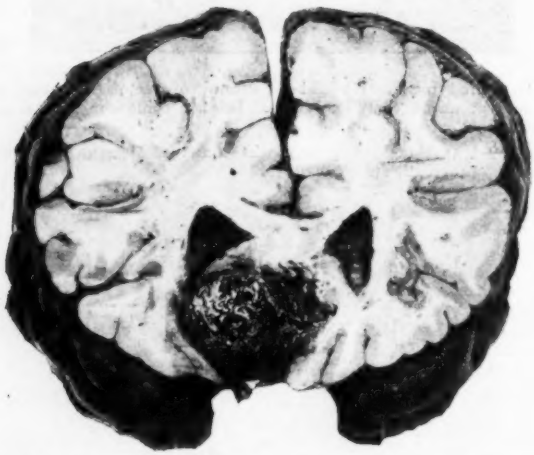

FIG. 4 (Case 1).—Position of vascular glioma (right frontal), whence hæmorrhage spread through the entire ventricular system.

approximately, of a period of Cheyne-Stokes' breathing for about one minute, associated with intense extensor rigidity of all limbs, inversion and hyperpronation of arms, flexion of wrists, thumbs between first and second fingers. The stiffening of the limbs preceded the onset of the altered breathing by a second or two; the face flushed slightly; the eyes always opened widely and the pupils dilated. In the feet there was usually flexion of the small toes, while the great toes were dorsiflexed proximally and flexed distally.

Death ensued after almost exactly seventy-two hours of complete decerebrate rigidity, with many scores of tonic fits. Post-mortem, a vascular glioma was found on the under-surface of the right frontal lobe (fig. 4), from which hæmorrhage had taken place, making its way right through the ventricular system and under the basal meninges, and spreading beyond the cranial cavity to the spinal theca. The third and fourth ventricles were blocked with blood.

The phenomena exhibited by the patient during these hours resemble in minute particulars those of mesencephalic transection in experimental animals. Until the hæmorrhage and increasing pressure killed him he was little or no more than a decerebrate preparation.

Case 2.—L. C., aged 34, a soldier, was admitted to hospital at 10 p.m. on the evening of January 29, 1918, with the history of having had a "stroke" earlier in the evening.

On admission he was conscious, and was able to speak fairly clearly. The left face, arm, and leg were not moved voluntarily. Complaint was made of severe pain in the head, which he frequently rubbed with his right hand. The pupils were of medium size, equal, and reacted to light. The mouth was drawn slightly to the right. All the deep reflexes were brisk, the knee-jerks excessively so, and there was double ankle clonus, with a double extensor-plantar response.

About 1 a.m. the patient suddenly became quite unconscious, and shortly after a series of convulsive attacks commenced and continued uninterruptedly for many hours, till within a short time of the patient's death the same day, January 30, at 2.25 p.m.

These attacks were of the following nature:—

The whole of the musculature of trunk, arms and legs became rigid; the arms and legs were extended to the fullest extent, the arms by the sides and the legs together. In addition, both arms were strongly inverted and hyperpronated, while the hands were clenched and the wrists slightly flexed. The legs were slightly inverted, the feet more notably so, with the toes pointing down and in. It was remarkable that notwithstanding the pre-existing paralysis on the left side, the musculature of both sides was equally and symmetrically involved in the tonic fits. While the musculature was in this rigid condition, fresh "waves," as it were, of tonic contraction passed through it, accentuating for the moment, if that were possible, the already existing posture.

The duration of each attack was less than half a minute. All through the night and far on into the next day these tonic fits continued, the patient never regaining consciousness. The interval between each was simply a few minutes.

At the autopsy recent fluid blood was found exuding from the pial roof of the fourth ventricle and spreading over the corpus callosum and over the whole of the basal surface of the hemispheres. On section an extensive recent hæmorrhage was seen to have ploughed up the interior of the right cerebral hemisphere and to have burst into the lateral ventricle, and so to the opposite lateral ventricle and right down the chain of ventricles to the fourth and through the foramina in its pial roof. The blood had also passed down the surface of the cord. There was obvious syphilitic disease of the cerebral vessels, and a small unruptured aneurism was found on the right middle cerebral artery. The free ruptured ends of the lenticulo-striate set of vessels on the right side lay in blood clot, but no aneurisms were found on them.

Case 3.—L. F., aged 39, married woman, was admitted to the National Hospital, under the care of Dr. James Taylor, on January 27, 1905.

In July, 1904, the patient began to suffer from severe headache and pain in the left occipito-parietal region, with giddiness.

In January, 1905, some weeks before admission, she noticed that her articulation was becoming defective, and within a few days the right arm and leg became distinctly weak, and the seat of occasional paræsthesiæ.

On her admission, moderate optic neuritis of both discs was found. There was well-marked weakness of the right side of the face, for both voluntary and involuntary movements, though less for the latter than the former. The patient lay in bed with the face turned to the left, the chin slightly up, the occiput to the right and rather down towards the right shoulder. The right arm was closely adducted to the chest, elbow at a right angle, forearm incompletely pronated, wrist extended, fingers and thumb slightly flexed. The whole limb was in a state of extreme hypertonus, and only slow and very weak voluntary movements were obtained in the muscle groups. The right leg was fully extended at hip, knee, and ankle, strongly hypertonic, and immobile.

A very brisk jaw-jerk was obtained, and though the patient did not complain in any way of the left limbs, and their volitional power seemed unimpaired, yet both abdominal reflexes were absent and the plantar response was definitely extensor on both sides.

On the fourth day after her admission a series of clinical phenomena of an unusual nature commenced and continued till her death five days later.

It was observed on the forenoon of January 31 that the patient was becoming drowsy and at one o'clock the left arm and leg suddenly passed into a rigid tonic spasm and relaxed again after five minutes. A similar tonic spasm of the left side occurred at two o'clock, of about the same duration. At six o'clock she was absolutely unconscious, the pupils widely dilated and immobile to bright light, respirations so shallow and noiseless as to be almost imperceptible, rate 16; pulse only 42, and slightly irregular. The patient lay immobile on her back, with limbs fully extended, and unexpectedly limp, only the slightest resistance to passive movement being noted in the joints. Within a brief period, however, respiration suddenly became noisy, irregular, and cogwheel or sobbing in character, and at the same time the extended limbs passed into a state of extreme rigidity in the decerebrate attitude; the upper extremities were adducted and internally rotated, the forearms strongly hyperpronated, so that the backs of the hands faced each other; trunk and neck were extremely rigid. and the latter slightly retracted; the legs were similarly in full extension, heels drawn up, toes pointing down and feet inverted. From time to time during the next few hours the rigidity waxed and waned, within moderate limits, but by 1.30 a.m. it ceased and the patient's limbs became once more relaxed, though the attitude of extension and pronation was unaltered.

During the next day the same alternation of tonic fits of all the muscles of the body with relaxation of tone occurred at intervals. That morning, when

the mouth was widely opened with a gag, it was seen that the uvula and soft palate were the seat of a rhythmical contraction and relaxation at the rate of about 120 a minute, entirely symmetrical on the two sides, there being no deviation of the raphe. This so-called "palatal nystagmus" continued without break or intermission for hours, and was re-observed at intervals all day. It is worthy of special attention.

On February 4 the limbs were flaccid and the patient apparently lifeless, respiration being imperceptible. Yet the extensor attitude never changed.

Death ensued the same day. At the post-mortem examination a tumour of the mesencephalon was discovered, invading the upper part of the pons. It proved to be a glioma.

Case 4.—M. B., a little girl, aged 6 years, was admitted to the National Hospital under my care on March 13, 1920.[1]

The history as given by the child's mother was that some weeks previously the patient had caught a cold, become rather deaf, and developed earache, which was followed a week later by headache, vomiting, and a rise of temperature. After three days of high fever her condition rather improved, but the headache and the vomiting persisted; actual otorrhœa never occurred. During the week previous to admission the symptoms became aggravated, and mild delirium set in, with restlessness and talkativeness. A squint also developed. The child was practically unconscious on admission to the hospital.

Neurological examination revealed hyperæmia and some blurring of the discs, and bulging of the tympanum of the right ear. Almost complete right and incomplete left ophthalmoplegia, interna and externa, with weakness of the left side of the face, was noted.

The child lay in a semi-conscious state with eyes half closed, mouth open, and head turned to the left. The left arm was flexed acutely at the elbow, adducted at the shoulder, and extended at the wrist, the fingers being flexed; the right arm lay in an easy extended position; the legs were fully extended and adducted, and the toes pointed downwards (fig. 5). From time to time the child moved her limbs in a vague and restless way, moaning the while, and occasionally opening her eyes.

At intervals during the twenty-four hours of her life which remained she exhibited tonic fits in a highly characteristic form. These were most commonly bilateral, but occasionally more severe on the left side than on the right.

A brief period of excitement and moaning ushered in the attacks, which consisted of sudden powerful opisthotonos and head retraction, the neck straightening and the occiput nearly touching the shoulders. The left arm relinquished its flexed attitude and became strongly extended, the forearm overpronated, the wrist extended to a complete right angle, the first and second fingers extended and the third and fourth flexed, while the thumb was flexed

[1] For the notes of this case I am indebted to my house physician, Dr. M. A. Blandy.

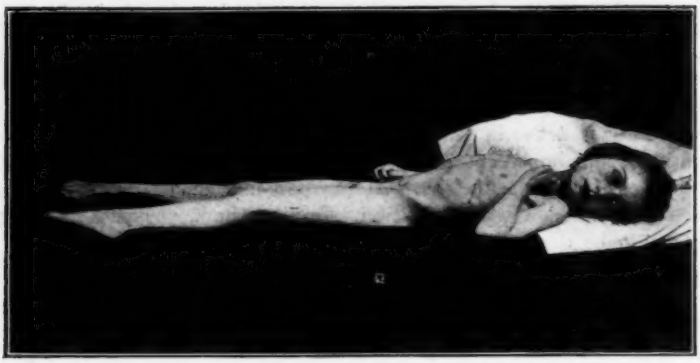

FIG. 5 (Case 4).—Incomplete decerebrate attitude in the intervals between the tonic fits. On the occurrence of the latter the left arm passed to full extension-pronation while the head straightened and was mesially retracted.

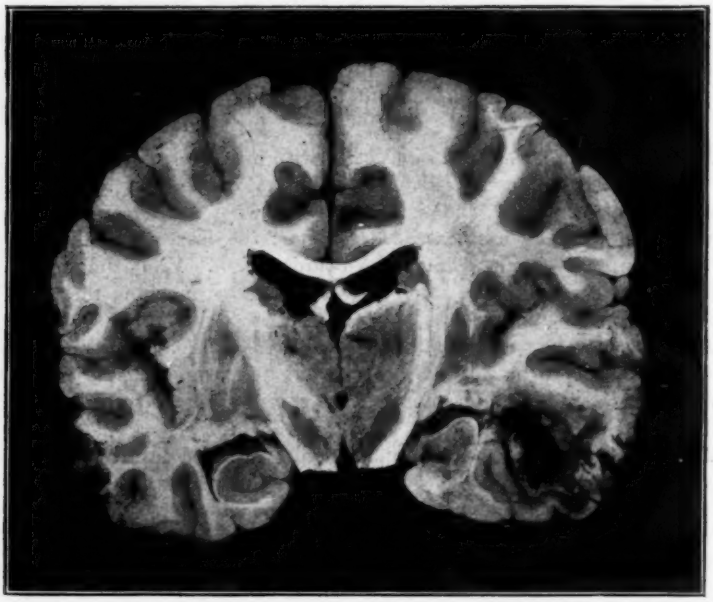

FIG. 6 (Case 4).—Shows position of temporal abscess from which the ventricular system became infected.

into the palm. The legs were in fullest extension with heels up and toes down, and the right arm was as the left, except that it was rather less inverted and the hand was in the form of a fist. Trunk and limbs alike were absolutely rigid. During the attacks, which lasted about ten to fifteen seconds, respiration quickened, and was accompanied by slight expiratory groaning. No clonic movements occurred at any time in the course of the attack, cessation of which was followed by relaxation of the hypertonicity and resumption of the original attitude described above.

Examination of the reflexes showed absence of the abdominals, activity of all the deep reflexes, bilateral extensor plantars, and a crossed extensor from the right to the left.

Lumbar puncture revealed a somewhat turbulent cerebrospinal fluid, in which organisms of the streptopneumococcus class were found. Death ensued within thirty-six hours of the patient's admission.

At the post-mortem examination there was general meningitis over the cortex and more marked along the base. The right middle ear was full of pus. Section revealed an abscess in the right temporosphenoidal lobe, whence, apparently, the ventricular system had become infected (fig. 6). There was but slight internal hydrocephalus.

Case 5.—M. C., aged $2\frac{3}{4}$ years, was admitted to the National Hospital under Dr. Ormerod on February 28, 1906.

Three weeks before admission the little girl had had a generalized epileptic attack, followed by another an hour or so later, Since, she had been apathetic yet irritable, occasionally giving vent to a cry. She had also become inattentive to her excretory functions.

On admission, the patient was drowsy but irritable, lying more or less constantly on her side with complete flexion of all her limbs, and with her back bent, so that she was literally "huddled up" in bed. The limbs could be passively extended at all joints, but the procedure brought out a certain amount of resistance in the flexors from hypertonus. The knee-jerks were active and the plantars flexor.

March 3, 1905: Patient comatose and roused with great difficulty. Occasional short spasmodic flexion movements of all limbs. Temperature 100·8° F.

10.30 p.m.: The arms remained flexed at all joints, but the legs had become fully extended at hip, knee, and ankle. Abdominal reflexes absent, knee-jerks active, double extensor response.

March 4, 1906: The extensor position of the legs was not maintained; they were sometimes flexed, sometimes extended.

March 6, 1906: The legs were now extended again, and so were the arms. The patient lay unconscious, with the arms extended by the sides and the forearms notably hyperpronated. The hands were clenched. The legs were also fully extended, with feet inverted and toes pointing down and in.

At frequent intervals tonic spasms passed down the musculature, re-

inforcing and accentuating the persisting extensor attitude of arms and legs. It was noteworthy that in these brief attacks the pronation of the forearms was always markedly increased. The trunk was extended, but not to an opisthotonic degree, nor was there more than slight retraction of the head.

This extensor rigidity, with occasional increase from transient extensor and pronation spasms, was maintained unchanged for the next few days, in fact up to the patient's death on March 11, 1906 (fig. 7).

Post-mortem: Typical tuberculous meningitis of the base, with moderate internal hydrocephalus.

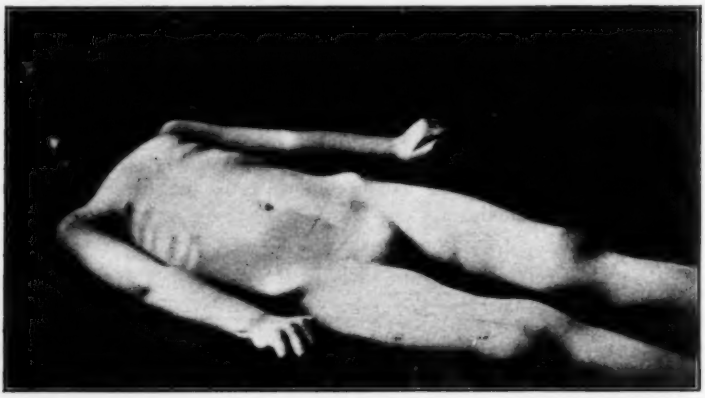

FIG. 7 (Case 5).—Decerebrate attitude maintained for several days *sub finem*.

A consideration of these five cases will suffice to illustrate the cardinal features of decerebrate rigidity in man, in its complete form. In each the decerebrate posture is reproduced with the exactness of a physiological experiment, and once it is appreciated it can always be recognized for what it is. The details of the attitude in man being on a larger scale, can be studied with greater ease than in mammals. One of its most significant components is the extension-pronation of the arms and the flexion of the wrists. We shall see later how it is just these components which are most likely to make their appearance in incomplete cases. The reader will also note that in all five examples the onset of tonic fits merely accentuated the already existing posture, showing that tonic fits are in reality attacks of decerebration, if the expression be allowed. The absence of any clonic element is of much significance. It may be taken to indicate that tonic and clonic movements have different origins and are the expression of the activity

of different motor mechanisms; one is characterized by phasic and the other by static activity.

From the pathological viewpoint it will already be apparent that in each instance the morbid process was so situated as to dissociate the cortex functionally from lower centres, which exhibit an activity of their own until such time as they themselves are overwhelmed.

It is particularly noteworthy that in many of the cases striking respiratory disorder ushered in the tonic attacks or persisted with the maintenance of the decerebrate posture.

(2) Cases of Decerebrate Attitude without Tonic Fits.

Case 6.—I owe this case to the kindness of my colleague at King's College Hospital, Dr. G. F. Still.

D. S., aged 15 months, was admitted to hospital, under the care of Dr. Still on February 28, 1920, with a history of "being ill" for six weeks. The chief clinical symptom was persistent vomiting after every meal for the previous three weeks. It was noted by the mother on the day of admission that the child's eyes seemed "fixed" and that its limbs were sometimes trembling and sometimes stiff. No fit had been observed, but for a day or more the infant was thought not to have recognized its mother.

On admission the optic discs were normal, the pupils reacted rather sluggishly to light, the fontanelle was flat. The child lay in an apparently unconscious condition with arms and legs extended and rigid, but without head-retraction. Kernig's sign was not obtained. From this date to the child's death, six days later, the attitude of decerebrate rigidity was maintained and indeed intensified. Thus the arms were fully extended, adducted, strongly hyperpronated; the backs of the hands faced each other, and at the same time the wrist and finger flexors were conspicuously contracted; the neck was stiff from muscular rigidity but was not retracted in any degree; the lower extremities were in full extension and adduction, with heels drawn up, toes pointing down, and feet slightly inverted (fig. 8). The abdominal reflexes could not be elicited, the knee-jerks were active and the plantars extensor. With a pulse of 150 was coupled a slow, sighing respiration. The child died quietly on March 9, after unconsciousness and decerebrate rigidity of some nine days duration.

Permission for a post-mortem examination was refused.

(3) Cases of Tonic Fits without Persisting Decerebrate Rigidity.

Less infrequent, possibly, than cases belonging to either of the two previous categories are those in which the occurrence of tonic fits is not associated with any maintenance of a decerebrate-rigidity attitude apart from what obtains during the fit itself. "Cerebellar fits," so-called—not

the best name to give them—have long been known, and since the days of Hughlings Jackson little has been added to his descriptions and interpretations. It is, however, desirable, not merely to bring the phenomena of tonic fits into line with what experimental physiology

FIG. 8 (Case 6).—Striking decerebrate posture of limbs and trunk, but with no head retraction. Photo taken from above, with child lying on back.

has taught us of decerebrate rigidity, but also to show that *it is possible to resolve these complete seizures into component elements, that partial, incomplete, even segmental tonic attacks may be met with, and that some at least of the more or less fixed attitudes of head and neck, trunk, and of one or other limb or part of a limb, encountered in certain basal, subtentorial and other lesions, are in reality functional fragments, so to say,*

of a non-cortical motor mechanism which finds its full expression in decerebrate rigidity and in complete tonic fits.

For the moment it is immaterial to our purpose whether these "tetanus-like seizures," as Jackson called them, are of organic or hysterical origin; a number of instances of each have come under observation.

Much the most striking of all is the following case, difficult though it be of exact diagnosis.

Case 7.—V. S., aged 18, was admitted to the National Hospital under the care of the late Dr. C. E. Beevor, on April 10, 1906.

Some months previously he had fallen down a flight of stairs on to his head, and though momentarily stunned and thereafter dazed for some hours he had not, apparently, really lost consciousness. Two weeks later he was thrown down some stairs, apparently in a "rag," struck his head again, and lost consciousness, but it is not known for how long. Thereafter he complained of pains in the head, shakiness, and drowsiness. Two months later he had an attack of influenza, and two days thereafter his first fit. These were of a peculiar character, described in detail below, and recurred on an average three or four times a week. They presented certain diagnostic difficulties, it is true, and opinion was divided as to their organic or hysterical nature, but it is important to note that the patient had had the attacks when in company and when alone in bed, indifferently, and that during his stay in hospital the efforts made to inhibit or cut short the attacks by the usual methods adopted in cases of hysteria were on each occasion a conspicuous failure.

On objective examination no signs of any organic disease of the central nervous system were found. The optic discs were clear and the reflexes in all respects normal.

The following is an account of a series of fits observed by myself.

When I reached the patient he was already lying in the semi-conscious condition succeeding a fit. He was breathing heavily, foaming slightly at the mouth, and was on his back. A moment later, he passed into a fit.

His arms went out in front of him, extended and hyperpronated, with hands clenched and wrists flexed. The eyes rolled up and the eyelids closed. The face appeared drawn, as if in a tonic spasm, while the arms and to a less extent the legs were absolutely stiff. Then the arms went out from the sides, and he began to rotate from left to right, so that he turned right round and lay on his face. This turning movement was slow and deliberate, and it was accompanied by slow stertorous respiration. At the same time as he turned his body became slightly curved anteroposteriorly, with the concavity forwards. He did not turn any further, but lay thus on his face. There was no clonic movement of any sort. Then in a few seconds came a relaxation, whereby the tonic spasm died down, and the limbs became completely atonic, flabby, so that they could be shaken about like flails. His respiration became more easy, and his colour less congested.

Nurse and I turned him round on to his back again.

A moment later, he passed into a second fit.

His corneal reflexes, tested at the commencement of this, were absent. The eyes deviated strongly upward, and at the same time the left arm went out into a tonic spasm, being away from his side and hyperpronated. At the same moment as his arm went out, he arched his body backwards, so that the occiput was down on the nape of the neck, and the muscles of the back in a powerful tonic contraction. The right arm was by the side, and it then contracted strongly in extension and internal rotation. With this, he again began to rotate, the left arm leading the way, and the trunk coming round after it from left to right, until he had come round on to his back again, nurse helping him, otherwise he would have been off the bed.

The same sequelæ, relaxation of the contractions and complete atonia, were observed. In the interim, the corneal reflexes returned, but he made no response to stimuli.

He then passed into a third fit.

This began by his rising off the bed completely, owing to strong contraction of the recti abdominis, &c., and in this anteroposterior plane he curved right forwards. Then his arms went away from the sides, the left before the right, both being powerfully rotated internally, and the hands clenched. The fists were strongly flexed on the forearms. The eyes were closed and the face drawn and quivering slightly. The under lip was curved, the corners of the mouth being depressed.

The position just mentioned was maintained for a moment, then he began to rotate slowly to the right, and at the same time to arch back into an opisthotonic position. A moment later, therefore, he was on his left side, his arms as before, his back most powerfully contracted, the head right back, the face looking upwards. Round he came still further, till he was on his face, and as he was breathing heavily we turned him on to his back again. There was no clonic movement whatever. I tried his jerks rapidly, and found they were all increased, the abdominals were diminished, especially on the left side, and there was a double flexor response. There was no incontinence and no biting of the tongue.

He sank a few seconds into the same atonic condition, with increased jerks, and lay motionless, salivating slightly.

Almost as soon as he had come round on his back the muscles relaxed and the limbs again became peculiarly atonic. He breathed deeply and his colour improved. There was no extensor response, but both abdominal reflexes were diminished.

A minute or two later, he put out both arms by his sides, separating his fingers, and then opened his eyes widely. His respiration quickened and he smacked his lips a little.

In this remarkable series of absolutely pure tonic fits, whatever their pathogenic basis, it is plain that the postures adopted were for the

major part precisely those of decerebration, especially in regard to the extensor rigidity with pronation of forearms and flexion of wrists, and the head retraction and opisthotonos. The changing nature of the movements, notably the occasional trunk flexion and the rotation, indicates that in persisting decerebrate rigidity the posture may be in part compounded of mutually antagonistic couples, which may be dissociated in the course of an actual tonic fit, and in part the result of one of these being for some reason overborne by the other. Reference is made to this consideration in a subsequent section. Where there is no persisting *structural* cortico-mesencephalic separation there is less likely to be fixity of posture.

Of several other cases of tonic fits associated with unmistakable organic disease the following may be selected as sufficiently illustrative.

Case 8.—The patient was a woman of 28 with a comparatively short history of headache, giddiness, vomiting, and optic neuritis. These general symptoms of intracranial tumour were not, however, accompanied by any definite localizing sign, although the persistent pain over the left side of the head warranted a left occipito-parietal decompression. During the five days between the operation and the patient's death she had several fits of a strictly tonic nature.

She suddenly became unconscious while the head wound was being dressed, both legs going out into strong extensor tonic spasm, the knees adducted and the feet powerfully plantar-flexed and inverted: the arms were similarly extended and adducted, with forearms hyperpronated on one occasion, but on another definitely rotated outwards, i.e., in a position of supination; the fingers were strongly adducted and extended, while the thumbs were flexed at the metacarpo-phalangeal joints and adducted across the palms. The head was markedly retracted and there was a distinct degree of opisthotonos of the spinal column. During the attacks, which lasted not more than 30 seconds, respiration always became very irregular. Immediately after the cessation of the tonic stage the limbs relaxed, seemed empty of tone, so to say, and could be moved about like flails.

At the post-mortem examination a glioma, of the size of a walnut, was found invading the cortex and white matter of the middle lobe of the cerebellum towards its left side, and of part of the left lateral lobe. From the operation wound and subcortical exploration then made blood had oozed into the left lateral ventricle and passed downwards.

It is obvious that the presence of the tumour alone will not explain the generalized bilateral tonic fits, and I attach more pathogenic importance to the results of the operative interference and the intraventricular hæmorrhage.

The occurrence of tonic fits as a hysterical manifestation is a neuro-

logical platitude, but their real significance for the purposes of this paper consists less in their psychogenic origin than in the morphological aspect they may assume. If we imagine the most superficial degree of hysterical dissociation to involve the transcortical neuronic level the movements released will be purposeful and complex, resembling normal motor activity except for full conscious participation therein; if, similarly, we assume functional disintegration to descend to lower neuronic levels we should expect to observe phenomena morphologically analogous to those of decerebrate rigidity, which is, as a fact, precisely the case. In this way the actual movements of certain moderately severe hysterical seizures receive a physiological explanation and illustrate the disorders of function which we have associated with organic decerebrate human preparations.

It seems unnecessary to cite any case in support of this contention, so commonly are the opisthotonic and extension-pronation postures found in hysteria. The reader is referred to one of Richer's drawings,[1] from which it will readily be seen that the attitude of the *arc de cercle* is strictly comparable to that following mesencephalic transection.

Scattered through the literature are a fair number of cases of what should be called "decerebrate rigidity in man," some of which have been observed with sufficient minuteness to be of documentary value. Of older cases, that recorded by Bastian [1], under the caption: "A Case of Apoplexy in a Boy, aged 15; Intraventricular Hæmorrhage, Convulsions, and Death in Four Hours," may be mentioned, and there are, of course, the original cases of Hughlings Jackson [11], some of which were republished in *Brain* (1906), and several to which he himself referred (Dreschfeld, Drummond, Bacon). One of the cerebellar cases of Stewart and Holmes [16] (No. 9 in their paper) is of particular value in this connection, and the recent article by Douglas Firth [6] may also be referred to. A good example of tonic fits in association with a large cyst of the cerebellum was reported by Eskridge [5]. Of particular interest are two papers by Bruce, one entitled: "A Case of Intracranial Aneurism in a Young Subject" [3], and the other, "Aneurism of the Anterior Cerebral Artery with unusual Prolongation of Life after Rupture; Autopsy" [4], and a third, published by Dr. Violet Turner [19], under the heading, "A Case of Prolonged Hyperpyrexia in a Child with a Mid-brain Tumour," of which three cases a précis follows.

[1] Paul Richer: "Etudes cliniques sur l'hystéro-épilepsie." Paris, 1881. Plate III, fig 2.

(1) *Bruce* (1904).

The patient was a miner, aged 27, whose chief complaint was of absolutely unbearable headache, but apart from slight congestion of the optic discs no objective neurological sign was discovered on minute examination. Ten days later he developed paresis of the right third and sixth cranial nerves. After temporary improvement, a fortnight from his admission the symptoms suddenly became acute again, and at 8.20 one evening he had a general epileptic fit, from which he never recovered consciousness. A second convulsion occurred at 9.15, and a third at 9.50. During the night numerous attacks of spasm occurred. For some hours these occurred about every five minutes, each period of spasm lasting about forty or fifty seconds. The spasm began with a gurgling sound in the throat; the breathing became accelerated (to 52 per minute) and noisy; and the pulse to 120 or 130. *The limbs became rigid: the arms were extended in front of the body and rotated so that the hands were placed back to back. The fingers were closed.*

The intercostal muscles and the diaphragm acted violently. In the intervals between the spasms the breathing was comparatively quiet and tranquil, and the limbs were placid. The pupils varied in size and reacted sluggishly to light. The patient died at 7 a.m.

At the post-mortem examination the base of the brain was covered by extensive diffuse subarachnoid hæmorrhage, spreading from the frontal region, optic chiasma, anterior surface of the pons and medulla, to the cerebellum. On opening the brain there was found a considerable quantity of recent coagulum in both lateral ventricles, but more abundant in the left. On separating the frontal lobes there was found, arising from the anterior communicating artery, a spherical aneurism about the size of a small hazel nut. It had burst on the left side and there was considerable softening and infiltration of the tissues of the left frontal lobe. The blood had passed both upwards into the ventricular system and into the subarachnoid space, where it had become widely diffused.

(2) *Bruce* (1908).

The patient was a smith, aged 33, who was admitted to hospital in a state of unconsciousness, which was said to have lasted about two hours.

On admission, he lay with arms and legs extended; the eyelids were half closed, the pupils contracted, circular, equal in size, and reacted to light only to a slight degree, if at all. Breathing was irregular both in rhythm and in depth of inspiration. The arms lay along the sides of the trunk, but *there were frequent spasms in which they were forcibly extended at the elbow and drawn to the side of the chest, or even in front of it. The forearms were very forcibly pronated, so that the backs of the hands were directed towards each other. The wrists were flexed, and the fingers were fully flexed, causing the hand to be tightly clenched, the thumbs being strongly adducted against the index fingers. During these tonic spasms the lower extremities were tonically extended, and*

there was extension at the ankle joints and inversion of the feet. The spasms lasted a few seconds, and were usually accompanied by a loud expiratory effort and puffing out of the cheeks, but the facial muscles were otherwise not involved. The head tended to rotate to the left side, and the chin was approximated to the left shoulder.

There was ankle clonus on both sides, but both plantar reflexes were flexor in type.

During the day the spasms continued. By 3.30 *the respirations were more definitely of a Cheyne-Stokes character, and at the height of the cycle the arms and legs became rigid and the back tended to be arched.* Lumbar puncture was performed, and 25 c.c. of a brownish red fluid were drawn off under high pressure. This fluid was obviously composed of blood and cerebro-spinal fluid mixed. By 8 p.m. the spasms had ceased.

The patient lingered in a comatose state for eleven more days, when he died.

At the post-mortem examination a small aneurism, the size of a pea, was discovered on the left anterior cerebral artery. It had given way, and from it the blood had spread directly into the subarachnoid space, passing along in all directions, filling up the sulci and the subarachnoid cisterns, to some extent "seeking an entrance" into the fourth ventricle from the foramen of Majendie, and also passing along the spinal cord under the arachnoid. The blood had also ploughed right through the corpus callosum and had entered the lateral ventricles and had coagulated there.

(3) *Turner* (1916).

The case was that of a female infant, aged 18 months, who was admitted to hospital in an unconscious state. Two months before admission the child had stopped talking and become dull and listless. For two weeks the legs had been stiff and crossed, and the pupils dilated. For the last three days the stiffness had increased, and opisthotonos had been noticed one day before admission.

On examination the child was quite unconscious, with half-open fixed eyes. The whole body was rigid, and the back arched with some head retraction. All four limbs were rigidly extended, and there was adductor spasm of the legs. All the deep reflexes were exaggerated; the plantar reflexes were extensor and the abdominal absent. After lumbar puncture and the removal of 2½ oz. of clear colourless fluid under great pressure the rigidity diminished gradually, and for one day the limbs were almost flaccid. The following day the spasticity returned and the fontanelle became very tense. From this time onwards until death, no less than twenty-eight days later, *the child lay in a state of extreme rigidity in the extended position, the feet in a position of marked plantar flexion, and the thighs adducted. The upper limbs were so rigid that at times they did not rest on the bed; the forearms were persistently pronated so that the palms were turned outwards, and the fingers flexed over the thumb,*

which was held across the palm. Actual opisthotonos was, however, only once present. The jaws were usually tightly clenched.

Mention may be made in passing of the extraordinary pyrexia in the case, reaching on one occasion 111° F. in the rectum, and 109° F. in the axilla.

Post-mortem, a solid rounded tumour was found situated in the mid-line in the region of the corpora quadrigemina (fig. 9). It did not involve the pons, cerebellum or optic thalami, but pressed forward into the aqueduct of Sylvius, causing a block, with resulting distension of the third and lateral ventricles. The tumour was apparently of a glio-sarcomatous type.

FIG. 9 (Turner's case).—The position of the tumour is such as to produce complete physiological decerebration at the level of the corpora quadrigemina.

These three cases in particular furnish ample corroboration of the connection between the peculiar symptomatology of decerebrate rigidity with postural fits and the neuronic level of the underlying lesions, and the identity of the postures in all confirms the support given by clinical observation to the findings of the physiological laboratory. The child in Turner's case was to all intents and purposes an experimental decerebrate preparation, and its survival for four weeks renders the case unique, as far as I have been able to discover, from this point of view.

It will doubtless occur to the reader that in various organic nervous conditions the phenomena under consideration make their appearance

in greater or less detail. Thus, not a few cases of Little's disease conform strictly to the decerebrate type. Attention may also be directed to the cases published by Sutherland and Paterson [17] under the heading. "A Type of Cerebral Mal-development (Forebrain Aplasia)." In these infants imperfect development of the cerebrum with relative conservation of the neuraxis below the basal ganglia has led, as might be expected, to the appearance of the decerebrate attitude, as can be seen by a glance at the figures in the paper.

Further, the familiar head-retraction of various forms of basal meningitis, with or without actual opisthotonos, is certainly but a part of the complete decerebrate posture. Reference will be made to this point again.

II.—(1) Cases of Decerebrate Attitude in Conscious Life.

There is no reason to doubt that the involuntary adoption, when consciousness is not in any way impaired, of an attitude corresponding exactly to the whole or any part of the decerebrate posture is to be attributed to the action of the same mechanism. The symptoms, it will readily be understood, are most apt to appear in circumstances

Fig 10.—Sir Charles Bell's drawing of a case of surgical tetanus.

under which the superior control of the corticospinal system is structurally or functionally defective; but, on the other hand, the characteristic postures are scarcely so likely to be maintained indefinitely as in the unconscious cases. There is, as it were, a conflict between two motor mechanisms, each attempting to jockey the other out of possession of the final common path.

I do not remember ever to have seen the assumption of the decere-

brate posture in hysteria during consciousness, though a case of this description is figured by Dejerine in the second edition of his "Sémiologie" (fig. 228, p. 544). In cases of tetanus the tonic spasms, consciousness being unclouded, are often closely analogous to those we have been considering, as Hughlings Jackson emphasized—hence his expression "tetanus-like seizures" for tonic fits, already alluded to. Fig. 10 is a reproduction of Sir Charles Bell's drawing of a case of surgical tetanus, observed by him on the retreat with Sir John Moore to Corunna. The flexion of the arms, in contradistinction to the usual extension of the typical decerebrate position, has, of course, frequently been noted in "cerebellar fits," and its meaning is discussed below.

FIG. 11 (Case 9).—Decerebrate attitude of upper limbs in a case of diplegia.

Cases of more or less Complete Decerebrate Attitude.

Case 9.—E. W., aged 2, was admitted to the National Hospital under the care of Dr. Risien Russell on March 1, 1920.

The child was a nine months' infant, born naturally, and breast-fed.

Apparently no particular notice was taken of her condition till she reached the age of six months, when her inability to sit up was remarked on. Her mother, nevertheless, admitted on being questioned that the child "had never used the arms properly" and that the legs tended to be stiff at the knees. The child had never walked.

On examination no impairment of function of the cranial nerves was detected. Intelligence, as far as could be ascertained, seemed good enough; body and head were well-proportioned. With support the patient was occasionally able to take a step or two in a very imperfect way, getting on to her toes and lifting her feet high. There was considerable spasticity in the legs and some varying rigidity of the trunk. Much the most noteworthy feature of the case, however, was the attitude of the arms, one not persistently maintained, but adopted involuntarily and interrupted only by voluntary movements. When the infant was lifted up or supported in a more or less vertical position the limb attitude was especially manifest, and it was often maintained for many minutes at a time (fig. 11). The arms were adducted at the shoulders and sometimes rather advanced, fully extended at the elbows and powerfully inverted and hyperpronated, with wrists extended and hands closed in flexion, presenting an absolute identity with the attitude of decerebrate rigidity already described.

Case 10.—H.O., aged 9, had been born at full time in a normal confinement. She was always weak in her arms and legs, and had not walked till she was three, and then very imperfectly. The history given by the parents was that she was slowly getting worse and that for six weeks she had not been able to walk at all.

When she came under observation her intelligence was noted to be very poor, and examination was rendered unusually difficult owing to her inattention.

What struck the observer was the extreme degree of volitional imperfection as regards movement: the child seemed to be unable to move any limb volitionally at all, and to succeed in doing anything to order only, as it were, by accident. The musculature of the body and limbs was distinctly rigid on passive movement, and the hypertonus was more noticeable proximally than distally. The abdominal reflexes were absent and the plantars extensor.

At one time the patient seemed to lie in a position of more or less generalized flexion, and at another, of generalized extension. In the former of these the trunk was bent forwards and the chin almost reached the chest; the arms were adducted, elbows flexed, forearms pronated, crossing each other on the chest; the thighs were flexed and adducted and the legs flexed at the knees. This huddled-up attitude seemed to be assumed mainly, though not solely, during sleep. When the patient was disturbed, or her limbs handled during an examination, she almost always passed into an extensor attitude (fig. 12), with head retracted, arms fully extended, inverted and hyperpronated to an

extreme degree: the legs were not, as a rule, fully extended at the hips or knees, though the feet were inverted, heels drawn up, and toes pointed down.

It should be mentioned that in addition to these attitudes the limbs were the seat of occasional slow spontaneous movements of an athetoid nature.

FIG. 12 (Case 10).—Extension-pronation attitude of upper limbs in a diplegic (see text).

(2) INCOMPLETE, LIMITED, OR FRAGMENTARY CASES.

Included in this section are cases where the decerebrate attitude is unilateral in arm and leg, or confined to one limb or segment of a limb. The more severe the degree of cortico-spinal interruption, corresponding to what obtains in actual decerebrate rigidity, the more likely is the unilateral attitude to resemble what is found in the complete cases, of which a number of instances have been furnished. It might be supposed, simply, that the ordinary hemiplegic attitude of arm and leg is a unilateral decerebrate posture, but this is the case only in a sense. One scarcely sees in experience the complete half attitude which would depend on the existence of complete unilateral cortico-spinal interruption, and this, owing to bilateral representation, cannot in fact occur. The upper extremity, again, assumes an attitude in flexion at the elbow, commonly, and not extension; nevertheless, the adduction at shoulder, pronation of forearm, and flexion of wrist and perhaps

fingers, are to be ascribed to the development, incomplete though it may be, of the decerebrate posture. It is important to recognize, in this connection, that in experimental unilateral decerebrate rigidity there are phenomena to be observed on *both* sides of the body. The following is Sherrington's [15] account of the condition:—

"If instead of both cerebral hemispheres one only, say the right, be ablated, the decerebrate rigidity appears, though not with the same certainty as after double ablation, chiefly on the same side as the hemisphere removed. The monkey, when slung after ablation of one—e.g., the right—hemisphere exhibits generally the following attitude. The right limbs are extended in the pose above described as characteristic for decerebrate rigidity, the tail is strongly incurvated toward the right, that is, its concavity is toward the right and its tip is also toward the right. It resists passive movement to the left, and if displaced thither immediately on being released flies back. The head also is pulled toward the right and retracted. The left forelimb . . . . is sometimes distinctly more flexed than would be expected in the paralysed condition of the animal; the left knee likewise. . . . . The contrast between the attitude of the crossed and homonymous sides is very striking . . . .

"Homonymous extensor rigidity consequent on ablation of one hemisphere is however neither so constant of production nor so persistent when it has appeared as the rigidity following bilateral ablation. After coming on it may totally subside and again reappear, and so on several times over. There would seem under these circumstances a struggle between two conflicting influences, as though a tonic influence from the still intact crossed hemisphere at times overcame and at times was overcome by another opposed influence from a lower centre. Some amount of extensor rigidity on the side opposite to the lesion is not uncommon."

Through the kindness of Dr. Poynton I am able to refer to a valuable case which he has afforded me every opportunity of examining, and which will serve to illustrate this part of our subject. The peculiar attitude presented by the child offers as close a resemblance to experimental unilateral decerebration as is likely to be noted in man.

Case 11.—J. R., aged 2, was admitted to the Hospital for Sick Children, Great Ormond Street, on February 21, 1920, under the care of Dr. Poynton.

The mother's pregnancy was normal, and the child was born at full time by easy labour.

It was suspected in his second or third month of life he was not a normal child, because he was so quiet and inactive. He "lay like a doll" in bed or in his mother's arms. He was quite conscious, and took the breast readily, but moved neither legs nor arms, neither cried nor smiled, and apparently took no notice of his surroundings. About the age of 6 months he began to use his right arm, moving it about in a jerky way, and to draw up and kick his legs about. Since then there had been a gradual increase in the movements of the limbs, especially on the right. He had never been able to hold his head

FIG. 13 (Case 11).—Attitude approximating to that of experimental unilateral decerebration. The photograph was taken from above, the child lying on his back on the floor.

up, sit up, or stand, and had never learned to talk. During the last six months restless and jerky movements of the limbs became rather more obvious. There was no history of convulsions or fits at any time.

On admission the child was seen to be a healthy-looking little boy, of almost normal weight and length for his age. He seemed intelligent and bright, and followed movements with his eyes, but did not understand the simplest spoken word. He cried if subjected to pain, and if hungry or interrupted during a meal, but did not smile or respond in any way to

pleasurable stimuli, except to cease crying if he were already crying. He made no attempt to speak.

There was no true paralysis in any limbs, since the arms and legs could be moved in any direction, but their power was very feeble, and volitional control seemed extraordinarily erratic. The boy was unable to sit up, stand, or crawl, and if placed on his belly he could not turn over. There was a constant tendency to adopt a peculiar attitude (fig. 13); when lying on his back he practically always kept the right arm level with the shoulder in an abducted position, and flexed to a right angle at the elbow, the forearm midway between pronation and supination, the wrist somewhat over-extended and the fingers and thumb flexed; the left arm, on the contrary, was usually fully extended and close by the side or slightly abducted, forearm pronated and hand closed in a fist. The right leg was flexed at hip and knee, everted and abducted, with heel drawn up and toes pointing down; the left leg, on the other hand, was, like the homolateral upper limb, extended in full and sometimes slightly abducted. As a rule the head was turned to the right and somewhat retracted, chin tilted up and occiput lowered.

There was moderate tonus of the musculature; neither hypotonia nor rigidity or contractures were present.

In addition, however, frequent involuntary movements of a more or less choreiform character, jerky and apparently purposeless (objectively, at least), interrupted the maintenance of the posture above described. These were best seen when the child was held up by the axillæ, the limbs moving jerkily and erratically as in a Punch and Judy figure, the trunk wriggling and twisting and the head rolling restlessly.

Sensibility to pain appeared normal. All the deep reflexes were active; the abdominals were present and the plantars of the flexor variety.

The Wassermann test was negative in the blood and cerebrospinal fluid.

When we pass to what I am convinced are in fact one-limb or even unisegmental instances of the same decerebrate state the subject becomes still more interesting. Little, if any, attention has been paid to this aspect of the question, significant and informative though it is, although in 1872 Hughlings Jackson made a passing allusion to it in a few words: "In some cases of cerebellar disease there is only a fragment of the tetanus-like condition."

If we take, for example, the question of the head alone, neurologists are familiar with what has long been known as the "cerebellar attitude" of that part of the body. It is characterized, in unilateral lesions, by a tendency of the head to be tilted back and the occiput to be turned to the side of the lesion (fig. 14). To recall Sherrington's words in unilateral decerebrate rigidity: "The head is pulled towards the right (in right-sided lesions) and retracted." Now in complete or bilateral cases

it is to be supposed that the *turning* elements to the right and the left respectively will neutralize each other, whereas the *retracting* element, common to both, will result in the accentuation of that posture. And this is precisely what is found in complete decerebration: the head is strongly retracted in the mesial plane, and rotation is absent. This is the common position in bilateral diffuse basal meningitis and other organic states. Should one (right or left) mechanism for some reason predominate over the other the lateral-tilting element will be reintroduced.

FIG. 14.—Illustrates the "cerebellar" attitude of the head in a case of right cerebellar tumour. The typical decerebrate attitude of the head is compounded of two partly opposing, partly reinforcing, "cerebellar" components.

A similar explanation holds good for curving of the trunk (spinal column). The opisthotonos of bilateral decerebrate rigidity is compact of right and left *extending* elements, reinforcing each other, whereas the right and the left *rotating* elements neutralize each other. Occasionally, however, in not quite complete cases, the lateral deviating element can be detected, as in the figures in Sutherland and Paterson's forebrain aplasia cases. It will be remembered, further, that in the tonic fits of Case 7 lateral-rotating mechanisms were in full activity during part of the seizures. Probably there is a similar dissociation of "corresponding opposites" in the phenomenon of skew deviation.

Though, as remarked above, such head and other attitudes are commonly spoken of as "cerebellar," it has been shown that they are in reality part and parcel of the postures of decerebration; hence the term "cerebellar" is doubly misleading, since it fails to denote this integral association, and since, as will be shown, it is doubtful whether the origin of the attitudes is cerebellar at all. Clinically, in not a few cases of head deviation that I have seen, the lesion has been mesencephalic and the cerebellum quite intact.

FIG. 15 (Case 12).—A fragment of decerebrate posture. The left arm shows the pronation phenomenon in a case of tumour of the left edge of the middle lobe of the cerebellum.

To pass, in the next place, to instances of the condition being confined almost to a segment of a limb, or even to one phase of segmental movement, particular attention may be directed to the "pronator sign," which is an important part of the complete decerebrate posture. Nothing is more characteristic than this pronation or overpronation; it was noted by Sherrington in his experimental animals, and we have seen the frequency with which it occurs in human cases of cortical "ablation." In certain cases of limited lesion dissociating cortico-spinal and mesencephalo-spinal function unilateral pronation of the forearm has been the sole definite and persisting sign, a small but integral fragment of the complete decerebrate attitude, and therefore of considerable clinical significance.

FIG. 16 (Case 12).—Photograph taken from behind the patient's head. The pronator sign in the left arm is an integral part of decerebrate posture.

FIG. 17.—Pronation of the left forearm in a case of cerebellar disease, presumably in the left of the middle obe.

Case 12.—E. L., aged 12, had suffered for one year from the usual symptoms of intracranial tumour, viz., headache, giddiness, vomiting, and was found on examination to have well-marked double optic neuritis. Almost the sole localizing sign was a somewhat indefinite reeling gait, pointing, however, on the whole, to a lesion in the left cerebellum. Accordingly, the patient was trephined over this area. A great part of the left lateral lobe of the cerebellum was removed under the impression that it was gliomatous, but eventually a quite small encapsuled tumour, about the size of a cherry, was discovered at the left edge of the middle lobe. During the operation pronounced skew-deviation made its appearance, the left eye being down and in, and the right eye up and out. When the patient was being tested some ten days after the operation it was noticed that the left arm, when extended, always assumed an attitude (figs. 15, 16) of marked pronation and inversion—a symptom which had not been present before, and in addition the left leg was held extended and inverted at the foot. Since the operation, further, the head definitely assumed the posture of occiput to the left, chin to the right. A month later these postural attitudes were still maintained.

Fig. 17 is from a case of cerebellar disease exhibiting the same phenomenon as in Case 12. The presumption is that the lesion was in an identical spot, viz., the left side of the middle lobe; no opportunity of corroborating this, however, was afforded.

(3) Cases of Partial Decerebrate Attitude occurring as Involuntary Movements in the Course of Chorea and Athetosis.

Finally, I have for a long time been impressed with the way in which this pronator sign, a fragment of decerebrate posture, shows itself in cases of involuntary movement, more particularly in chorea and athetosis.

When the patient with acute or subacute Sydenham's chorea is asked to extend his arms above his head, palms to the front, it is extremely common to find them adopting involuntarily an attitude of pronation, with the palms turned outwards. The movement is well seen by contrast in cases of hemichorea (fig. 18), and is sometimes found persisting after the actual choreic movements have ceased. It is also to be observed in so-called paralytic chorea. Thus through the changing foreground of choreic twitching can be recognized a postural background of mesencephalic significance.

Similarly, in athetosis the pronator attitude is a position of election; it is, for example, vastly more frequent than any attitude of supination. Difficult though it be to follow the changing movements of athetosis,

FIG. 18.—Well marked pronator sign in a case of left unilateral Sydenham's chorea (hemichorea).

FIG. 19.—Characteristic transient decerebrate posture of right upper limb in a case of diplegia with athetosis.

there is, in my opinion, no doubt that the substantial background of that condition is one of varying decerebrate posture. Only one case need be cited in support of this contention; others are sufficiently illustrated in the photographs (figs. 19, 20).

FIG. 20.—Case of infantile cerebral hemiplegia with athetosis (left). Extreme hyper-pronation and characteristic decerebrate posture, with the exception that the fingers are extended and not flexed.

Case 13.—A. W., aged 10, developed diphtheria in January, 1919. The attack was of moderate severity. About fourteen days after the commencement of the illness, and while the patient was still very ill, she developed a complete left hemiplegia, involving face, arm and leg, without loss of consciousness and without any fit. Some weeks after the attack it was observed that the left arm was the seat of slow involuntary movements of an athetoid nature.

When the patient came under observation in June 1919, the hemiplegia was noted to be slight in degree, implicating mainly the left face and the left arm. Though the leg was not much complained of an extensor response was present. The left arm showed fair muscular power, though weaker than the right, and a fair range of voluntary movements, but these were interrupted by characteristic involuntary athetoid movements, more especially of the forearm, hand and fingers. There was a constant tendency for these movements to place the forearm in the attitude of over-pronation (fig. 21). Under treatment steady improvement took place, till at present the athetosis has very largely

disappeared; but the pronation-posture, though less frequently assumed, is still to be observed, especially if the patient is asked to extend her arms above her head.

In addition to the pronator sign it is possible to pick out others of the postural manifestations during the maintenance of decerebrate rigidity and observe their reproduction in the course of chorea and athetosis. One more phenomenon may be alluded to in this respect. It will have been noticed that in the great majority of the complete cases the arms are flexed at the wrists; Sherrington's experimental

FIG. 21 (Case 13).—Slight left hemiplegia with athetosis, following diphtheria. The left arm adopts an involuntary position of over-pronation.

animals showed the same flexion. Such wrist-flexion is, of course, the usual attitude at that joint in hemiplegia, representing, as has already been said, a fragment of decerebrate posture, and precisely the same flexion is extremely common in the choreic hand (fig. 22); it is always well seen when the arms are voluntarily extended in front. The choreic hand has long been known, but its significance in connection with decerebration does not appear to have been observed. Coupled with the flexion of the wrists is a tendency to finger over-

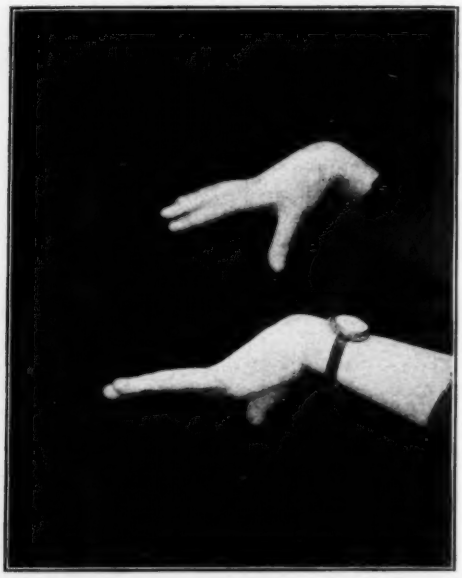

FIG. 22.—The choreic hand ; wrist flexion and some over-extension at the knuckles.

FIG. 23.—The athetoid hand, practically identical with the choreic hand (fig. 22).

extension, which is not observed generally in decerebrate rigidity (though in Case 8 the fingers were extended and adducted), but which, it must be understood, is largely voluntary in the test as usually employed, the patient being asked to "hold his hands out in front." This same flexion of the wrists is one of the commonest attitudes of athetosis (fig. 23).

There has recently been under my care at the National Hospital a case in which this combined wrist-flexion and finger-extension was a constant phenomenon in a child whose physiological condition was one not far from complete decerebration, and in whom the vestibular reflexes of Magnus and De Kleijn [14] were readily obtainable.

FIG. 24 (Case 14).—Microcephaly with diplegia and athetoid attitudes. For significance see text.

Case 14.—R. G., aged 3½, was admitted to the National Hospital under my care on June 5, 1919.

The infant was born after long labour with instrumental delivery. He was always backward, never tried to crawl, and could not use his arms properly. He never could hold his head up as other children.

On admission the child was noted to have a somewhat microcephalic head. He was extremely backward and infantile, though well enough developed

physically. Little if any attention seemed to be paid to interest taken in him. He wailed in a monotonous voice most of the time when left alone, but usually stopped when addressed, and occasionally an infantile smile crossed his features. The more or less constant attitude of the child is seen in fig. 24.

Magnus and De Kleijn reflexes.—When the child was placed on its back and the head gently turned, say to the left, and when it stopped crying, it lay quietly with upper limbs in a moderate flexion posture. On turning its head sharply to the right passively an immediate extension of the left arm into the usual decerebrate attitude was always obtained.

FIG. 25.—Torsion-spasm. The right arm is in a characteristic decerebrate posture.

One other variety of involuntary movement may be alluded to. In the rare disease known as torsion-spasm or torsion-dystonia (dystonia muscularum deformans) one or other limbs may adopt the decerebrate posture in its entirety. A striking case of this description under the care of my colleague Dr. Collier, who has given me every opportunity of examining it, has been fully reported by Dr. M. A. Blandy [2] and a photograph of the boy is here reproduced (fig. 25).

To recapitulate, thus far, in a sentence or two, the general view taken is as follows:—

(1) The symptoms of acute cases of physiological decerebration in men are identical with what obtains in animal experimentation. The decerebrate condition of forearm, wrist and fingers—extension at elbow,

pronation, flexion of wrist and usually of fingers—is commonly more marked than in animals, sometimes to an extreme degree. It is particularly noteworthy that respiratory disturbance often precedes or accompanies the decerebrate rigidity. The condition is frequently combined with tonic or postural fits, the feature of which is that they accentuate or reinforce the existing decerebrate attitude.

(2) Tonic or postural fits are, in reality, the transient adoption of this decerebrate attitude, complete or incomplete as the case may be. In the case of the fits there are changing phases of tonic attitude or posture which are less likely to be observed in the greater fixity of decerebrate posture produced by the release of certain neural mechanisms in structural nervous disease which *permanently* dissociate cortex from mesencephalon.

(3) The complete decerebrate attitude is compounded of unilateral elements which reinforce each other and of other unilateral elements which neutralize or antagonize each other. In the case of head and trunk postures, at least, it is possible to resolve them into components which, in incomplete cases, may preponderate on one or other side, producing deviations from the complete posture. This complete decerebrate posture is essentially composed of movements grouped in action in the mesial plane of the body.

(4) In cases where consciousness is intact and the corticospinal system partially or entirely in functional abeyance, portions of the decerebrate attitude make their appearance in the form of head, trunk, or limb deviations and attitudes; there may be fragments confined to one limb or segment of a limb. This is in particular the case with the pronator sign.

(5) In involuntary movements of the nature of chorea and athetosis it can be shown that there is a postural background which is composed of part of the complete decerebrate attitude. In both of these varieties the commonest phases are pronation and wrist flexion; the choreic and the athetoid hands are identical in this respect, and pronation and wrist flexion are an integral part of decerebrate posture.

(6) There is an important difference between the unconscious and the conscious cases, seeing that in the latter, unless the corticospinal interruption is absolute, the cortex may and often does interfere in part with the activity of the released lower mechanisms. In Sherrington's words, "there is a struggle between two conflicting influences."

The Pathogenesis of Decerebrate Rigidity.

We have seen that the resemblance between human and animal decerebrate rigidity is of the closest, and pathological evidence has been adduced indicating the nature and localization of the morbid processes likely to bring it about. From the type of pathological process, however, herein described as producing the syndrome (tumours, cerebral hæmorrhage, meningitis and internal hydrocephalus), it is apparent the localization can only be approximate, and physiological rather than anatomical; with other words, any lesion dissociating the cortex from the mesencephalo-cerebellar level and at the same time leaving the latter functionally intact, is calculated to cause the rigidity to develop, but the finer anatomical details can only be supplied at present from the experimental side. At the same time attention may again be directed to the fact that in the acute human cases marked disturbance of respiration has been repeatedly observed (see the cases in the first group, above). Graham Brown [10] has shown by exact experimentation that there is a respiratory tract, localizable with extreme nicety, in the mid-brain. In the ape it lies about 3 mm. from the aqueduct, and its stimulation produces gasping or panting movements. The occurrence of respiratory difficulty in the acute human cases may, perhaps, be attributed to interference with the function of this tract (assuming its presence in man) and thus provides important collateral evidence suggesting that the vicinity of the nucleus ruber (as is shown below), and not the cerebellum, is the neighbourhood whence is derived the activity that produces decerebrate rigidity.

The general physiological view enunciated by Hughlings Jackson [12] in 1877 as to the cause of hemiplegic rigidity is the starting point in our investigations of the pathogenesis of the symptom-complex under consideration. The following is his outline of the basic features of the "co-operation of antagonism" between cerebrum and cerebellum.

"There is evidently a duplex symptomatic condition, negative and positive; with loss of power over the muscles there is tonic action of them. Negative states of the nervous centres cannot *cause* positive states of muscles, they may *permit* them. My speculation is that the rigidity is owing to unantagonized influence of the cerebellum. . . . The cerebellum is the centre for continuous movements and the cerebrum for changing movements. . . . The former are tonic, the latter clonic. . . . In walking, the cerebellum preserves the equilibrium of the body, tends to "stiffen" all the muscles; the changing move-

ments of walking are the result of cerebral discharges overcoming in a particular and orderly way the otherwise continuous cerebellar influence. . . .

"When, however, the influence of the cerebrum is permanently taken off by disease of the cerebrum, as in hemiplegia, from the parts which it most specially governs, the cerebellar influence is no longer antagonized; there is unimpeded cerebellar influx, and hence rigidity of the muscles which in health the cerebrum chiefly innervates. The principle, I think, accounts for the duplex, negative and positive conditions in the contractures of hemiplegic children, in *the athetosis of Hammond*, and accounts for the rigidity and "propulsive" walk in paralysis agitans; temporary removal also for tetany and *certain hysterical attacks*, and also for some post-epileptic conditions of movements" (italics mine).

Specially noteworthy in this account is Jackson's recognition of "cerebellar influx" both in athetosis and in certain hysterical attacks; with characteristic prescience he sketched a hypothesis half a century ago, many of the details of which are only now receiving complete confirmation.

In regard to the above theory, however, the chief comment to be made is that the speculation as to the actual site of origin of the rigidity-producing influence being in the cerebellum cannot be considered proved, though the latter organ plays an important part. There is no question that the phenomena of decerebrate rigidity are release-phenomena, and that, again in Jackson's words, a lower influx invades what the higher gradually abandons.

For the determination of further essential facts in connexion with the subject of pathogenesis we must turn to the work of the experimentalists, conflicting though it is in some respects and not always of simplicity in interpretation.

In the first place, there is general agreement that decerebrate rigidity makes its appearance after transection of the neuraxis at the level of the anterior colliculi, and disappears by a second section below the neuronic level of the medulla. For its development, therefore, we are limited to the mesencephalon, cerebellum, and pons. It must, nevertheless, be mentioned there is experimental evidence suggesting that similar attitudes can be obtained when the decerebration is higher, leaving intact the basal ganglia, in particular the optic thalamus, and that movements analogous to those of tonic fits can be produced by stimulation of the latter structure (Lowenthal and Horsley [13],

Thiele [18], Graham Brown [8]). Rigidity, however, sets in with precision from mesencephalic transection, and for the moment we will confine consideration to the three parts of the neuraxis just specified.

This mesencephalic decerebrate rigidity, once established, can be abolished by various procedures. It will be readily understood that integrity of the lowest tone-effecting spinal proprioceptive arcs is assumed in this connection; with rigidity at its height, section of afferent roots at once abolishes rigidity for the limb concerned. Ordinarily speaking, therefore, for rigidity to appear a normal neuraxis below the mesencephalic level is presupposed, and if, clinically, a hæmorrhage spreads down the cord to invade local proprioceptive arcs the phenomena are certain to vanish. Thus the attitude of decerebrate rigidity may only be a phase in the course of a case and may disappear *sub finem*. Graham Brown [7], however, has made the important observation that integrity of these lower tone-effecting arcs is *not* absolutely essential. "The reactions (of decerebrate attitude) may occur many months after division of the dorsal spinal roots of the arm. That is to say, appropriate stimulation in the region of the midbrain may evoke an extensor postural tonus or a flexor postural tonus. Sherrington has found that the decerebrate rigidity which occurs after removal of the cerebrum does not occur in a 'de-afferented' limb, but the fact that a condition which at any rate very closely resembles this state may be evoked in such limbs seems to point to the conclusion that the absence of this postural tonus in the decerebrate 'de-afferented' animal is due to the failure of the ascending impulses from the limb which normally play—however indirectly—upon these mechanisms of the midbrain, and that the mechanisms themselves if properly activated are still able to induce the tonus."

In addition to the local spinal reflex arcs for tone, it is held by Sherrington, as Graham Brown says in the above citation, that impulses ascend to the midbrain mechanisms to maintain them in activity, but they do not pass by the dorsal columns, for section of these has no effect, whereas section of the ventro-lateral cord abolishes the rigidity. This suggests the conveyance of maintenance-impulses by the spino-cerebellar tracts, and Weed [20] believes the path is the ventral spinocerebellar tract of Gowers, and not the dorsal, since section of the inferior cerebellar peduncles, by which the dorsal pass to the cerebellum, is without influence on established rigidity.

Another method of abolishing the extensor tonus after decerebration is by excitation of the anterior (superior) surface of the cerebellum,

from near the midline outwards to near the lateral border of the cerebellar surface (superior vermis and outward). This has been observed repeatedly by Sherrington, Lowenthal and Horsley, Weed and others. If, then, stimulation of this part of the cerebellum inhibits an induced decerebrate rigidity, it might be supposed, conversely, that destruction of the same area would produce the condition, mesencephalic decerebration not being performed, and this has been done by Sherrington and by Thiele. Bisection of or injury to the vermis, median section of the cerebellum, and partial ablation of the cerebellum, are all apt to produce the phenomena under discussion, but Sherrington is not convinced that the two conditions are identical, and specially states that after these cerebellar operations decerebrate rigidity "sets in often, *but not always*" (italics mine). He has seen the condition "persist after removal of the cerebellum, if the latter ablation be performed without any serious amount of hæmorrhage." Graham Brown, similarly, has clearly shown that the reactions of decerebrate rigidity may be obtained from stimulation of the midbrain "after the complete removal of the cerebellum"; and Thiele has removed the whole cerebellum down to the paracerebellar nuclei (Deiters) without affecting the decerebrate posture.

It is apparent, therefore, that functional integrity of the cerebellum is *not* a *conditio sine quâ non* for the appearance of the syndrome after mesencephalic transection in animals, and that the phenomena may develop in the actual absence of that organ.

These experimental findings seem to render an actual cerebellar origin for the rigidities improbable.

The position reached by Sherrington, Weed, and Graham Brown is that the mesencephalon itself is of prime importance. According to Weed, the real origin of decerebrate rigidity is in the midbrain in the region of the colliculi. "In this portion of the midbrain, lying ventral to the colliculi, the tegmental structure which apparently affords origin for the rigidities is the nucleus ruber." Graham Brown has developed the hypothesis in greater detail and with a wealth of experimental material. He has shown that "the red nucleus is the centre the activity of which conditions" the postural reflexes constituting decerebrate rigidity. And in a personal communication to the writer Professor Sherrington says:—

"The neighbourhood of the iter taken broadly so as to include the red nucleus, small-celled and large-celled parts, certainly has large postural (tonic) functions, and Graham Brown's experiments observed those in detail. Work on decerebrate rigidity has gradually come to

suggest that the red nucleus region is of capital importance for the general reflex of 'standing' ('standing' in its various forms and sub-postures). Tonic (posture) fits would, to judge from laboratory observations, be likely to result from lesions of an acute kind in that region."

From the point of view of experimentation, then, the conclusion seems clear enough that the withdrawal of cortical control, by decerebration, from the spinal motor centres allows them to be played on by a non-pyramidal motor influence of a tonic nature, or rather allows this doubtlessly constant tonizing factor to reveal itself in action. The centre of this attitude- or posture-producing motor activity is apparently the nucleus ruber of the mesencephalon, and not the cerebellar cortex or cerebellar nuclei, and it is maintained in tone itself, ordinarily, by afferent impulses reaching it, "however indirectly," from lower (spinal) neural levels, though its activities are not necessarily paralysed by their absence.

The nucleus ruber, however, is no isolated and autonomous ganglion, but has numerous and important connections with structures above, beside, and below it. Further, and of equal importance, it is a twin nucleus. We cannot suppose, though it be the originator of decerebrate rigidity, that it is uninfluenced by stimuli from parts with which it is in close relation.

(1) Though the postural discharges of decerebrate attitude have not their origin in the cerebellum, the latter must certainly play a part, as long, that is, as it is in functional connection with the midbrain. We have already seen that the rigidity can be inhibited by excitation of the neighbourhood of the vermis, while lesions there would appear themselves to be provocative of rigidity, the mesencephalon being intact, or to accentuate existing rigidity. This experimental finding is supported by the clinical evidence of Case 12 above, since the destructive lesion allowing the "fragments" of decerebrate posture (pronation) to appear was situated at the left edge of the middle lobe, i.e., in the position whence decerebrate rigidity can be produced experimentally by destructive lesions. Thus the cerebellum would appear to exercise both an inhibitory and an excitatory action on the mesencephalic mechanism which is responsible for decerebrate posture. According to Weed, who produced the condition by removal of the cerebral hemispheres, leaving the internal capsules and basal ganglia (if I understand his descriptions correctly), it is possible to trace an inhibitory pathway from the cerebrum which corresponds to the fronto-

ponto-cerebellar route (viâ the middle cerebellar peduncle). Stimulation of this pathway always inhibited existing decerebrate posture, mainly homolaterally, suggesting a doubly crossed connection. Weed found the path is distributed, in part at least, to the cortex of the anterior portion of the superior vermis, whence, as has been said, previous experimenters have been able to inhibit the rigidity, but whether the inhibitory effect is exercised upon the basal cerebellar nuclei (Deiters) or upon the mesencephalic nuclei is not at present known.

As far, then, as cerebellar action is concerned, it may be tentatively concluded that (1) decerebrate attitude of mesencephalic origin is maintained by afferents reaching its source by spinocerebellar paths, and (2) inhibited by stimulation of a ponto-cerebellar path viâ the neighbourhood of the vermis, i.e., of what constitutes largely the palæocerebellum. Possibly the maintaining influence reaches the twin red nuclei by the superior cerebellar peduncles, since their section, in the decerebrate preparation, is usually followed by disappearance of the rigidity; as already noted, it is not certain how the inhibiting influence is conveyed to the mesencephalon, or even whether it may not pass back again to spinal centres by another route.

(2) The basal ganglia have intimate connections with the red nucleus. In particular, the corpus striatum is in relation with it by means of the ansa lenticularis. As our subject is mainly concerned with the phenomena appearing after transection through the midbrain, when the basal ganglia are out of action, it is not necessary to discuss the physiological connections of the two in this place, though several allusions have already been made to the fact that with suitable experimentation the rubral mechanism of posture can be activated from above, from the basal ganglia. In a subsequent communication concerned with the pathogenesis of involuntary movements further opportunity will be taken to analyse the bearing of the influence of the basal ganglia on mesencephalic function, and a brief reference to the connection of the red nucleus with tremor is given below.

We may turn next to the question of the rigidity-producing mechanism itself.

The careful work of Graham Brown has shown that in the decerebrate preparation electrical stimulation of certain points in the cross-section of the midbrain at the level of the anterior colliculi in monkeys yields characteristic reactions. The areas excited, his "focal points," lie on either side of the midline some millimetres ventral to the aqueduct

of Sylvius, and correspond with the anatomical position of the red nucleus (and possibly, also, with that of the posterior longitudinal bundle). "Stimulation applied to this area of the cross-section of the midbrain on one side evokes a state of flexion in the arm of the same side and a state of extension in the arm of the opposite side. On cessation of stimulation these reactions are continued as maintained postures. The movements of the arms are accompanied by movements of other parts of the body. The tail seems to bend to the same side; the lower limb of the same side extends while that of the opposite side flexes; the head is rotated in such a manner that the face is turned away from the side stimulated." He has shown, further, the mutual antagonism of the right and left focal points, and by immediate or successive compounding of the two has produced bilateral extension, bilateral flexion, and so on. "With varying values of stimulation varying values of contralateral extension and ipsilateral flexion might compound to give reactions in which extension and flexion exhibited differing preponderancies."

Now our clinical study has already demonstrated the fact that the clinical phenomena of decerebrate rigidity are compounded of elements some of which are mutually reinforcing and others mutually antagonistic, and in Graham Brown's work we have experimental proof of the way in which these symptoms are produced. It is evident that the varieties and alternations of positions—e.g., all limbs extended, as is usual in human decerebrate rigidity, or arms flexed and legs extended as in Jackson's "tetanus-like seizures"—are due to compounding of varying influences affecting the red nuclei or originating in them. And it seems no long step to the supposition that unisegmental attitude, as, for instance, in the pronator sign, is the result of limited or partial red-nuclear activity.

It may be suggested that in the red nucleus, perhaps in the pars magnocellularis, are represented combinations of the ventral cell-groups of the spinal cord, linked up according to their flexor, extensor, pronator, supinator, &c., functions. For we cannot believe that in the red nucleus we have other than a "re-presentation" of the ventral cord cell groups. Graham Brown has, indeed, demonstrated that the point of common antagonism between the twin nuclei is below the midbrain, and the natural surmise is that it lies at as low a level as that of the spinal centres. If this be the case, then stimulation of a point in the red nucleus, after decerebration, may be supposed to innervate a physiological group of ventral cells, the flexors, say, scattered throughout the

various spinal segments, and all linked to a nodal cellular representative in the former nucleus. The same must be true of extensor cells, &c. Uncontrolled activity of the twin nuclei from decerebration throws the individual into the decerebrate posture, with predominance or changing activity of flexor or extensor attitude on one or other side, in one or other limb, as the case may be, owing to changing influences in the red nuclei or reaching them.

Undoubtedly the prèdominating activity of the uncontrolled nuclei is one causing extensor tonus. The "neural balance" of a limb-pair, taken together, seems for some reason to be dominantly weighted or disposed towards, or "set in," extension [9]. Sherrington's explanation of this is that decerebrate posture is in reality "reflex standing," hence the preponderance of extensor tone.

Throughout this discussion, for the sake of clearness, the assumption has to some extent been made that it is precisely the red nucleus in which these postural activities are anatomically centred, though greater certainty is necessary before the matter can be regarded as settled. But it seems desirable to point out that the nucleus ruber is but a part of Edinger's nucleus motorius tegmenti, which consists of associated motor-cell groups scattered through the pons and mesencephalon to the level of the ventral optic thalamus, including Deiters' nucleus, and that the effect of stimulation is not likely to be limited to one descending rubral path, viz., the rubrospinal. There is, in fact, doubt as to the path or paths by which rubral activities reach the cell-groups of the ventral cornua; further, rubro-pontine and rubro-bulbar connections must not be ignored. In this connection it is well to mention that Thiele [18] attributes the conduction of decerebrate impulses spinalwards, not to the nucleus ruber and its connections, but to Deiters' nucleus, another part of the nucleus motorius tegmenti, and to the vestibulospinal tract. Thus there is room for further research and elucidation in the matter. It is clear, for example, that other influences reach the final common path of the lower motor neuron than the rubrospinal. The possibility of labyrinthine agency contributing a quota to decerebrate rigidity, originally mooted by Ewald, has been largely supported recently by the researches of Magnus and de Kleijn [14], who have proved that in the decerebrate animal passive movements of the head give rise to alterations of tonus in the limbs, and that each movement of the head varies in its influence on tonus according to the position of the animal in space. Only this passing allusion to their investigations can be now made.

Although the question is one to be dealt with elsewhere, brief

mention may be made, in conclusion, of the connection of the red nucleus with involuntary movement. If the hypothesis suggested above be tenable, viz., that the flexor, extensor, &c., groups of the spinal cells are respectively linked to nodal representatives in the nucleus—are, as it were, re-assembled in these twin ganglia—then we can understand how tremor may occur in association with rubral disorder. For, in Jackson's words once more, "tremor differs from rigidity, not fundamentally, but in degree."

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RELATION OF THE FORM OF THE KNEE-JERK AND PATELLAR CLONUS TO MUSCLE TONUS.[1]

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I.—Introduction.

Variations in the form of the knee-jerk under various degrees of muscle tonus have been observed both clinically and in the physiological laboratory. These observations have extended over many years from the clinical deductions of Erb and Westphal in 1875, through the more recent physiological experiments of Sherrington and others. From these studies the clinician has drawn certain practical concepts such as "exaggerated knee-jerks," "spastic knee-jerks" and "spasticity," terms used to denote the stiffness of the muscle and the increase in the amplitude of the reflex response most commonly seen after lesions of the pyramidal tracts. These expressions are used in a rather loose way, simply denoting gross quantitative changes, for the most part not to be measured with much degree of accuracy.

Attempts, however, have been made to measure and record variations of the knee-jerk in man and to show the relations between these variations and muscle tonus. Weir Mitchell and Lewis [11] in 1886 contributed the first of such observations and came to the conclusion that "the responsive jerk brought about by striking a stretched tendon is the most refined means we possess of deciding as to the tone of a muscle." This, I think, has been borne out by research since that date, although we have developed more "refined means" to aid in the decision. Bowditch and Warren [1] in the Harvard Laboratory two years later measured the knee-jerk in man by a cleverly arranged but complicated apparatus. Lombard [4] also carried out experiments on man. Somewhat later Wertheim Salomonson [12] published a paper on reflex studies in man with kymographic records. It was noted by all experimenters on men that numerous difficulties prevented the correct recording of their observations. Most of these workers found great mechanical difficulty in attaching a steady apparatus to the lower

[1] From the Physiological Laboratory, Oxford University.

extremity. Voluntary movements of the patient or his voluntary inhibition of the reflex also caused great variations in their result. These latter phenomena could not be successfully controlled. In short, all attempts to study any single phase of the knee-jerk in man, such as its form, and to record the observations graphically have been complicated by so many factors which could not be controlled that results from such studies are only partially satisfactory.

In animal experiments, however, many of the extraneous factors mentioned above can be controlled by the experimenter. Unfortunately, except for the work of Sherrington, there have been few detailed studies in animals on the relation of the form of the knee-jerk to muscle tonus. It was the aim of this research, then, to demonstrate in animals just what form the knee-jerk may assume under various degrees of tonus. We only attempt to isolate the knee-jerk nerve-muscle group in a living preparation and study it under various degrees of muscle tonus.

II.—Method.

(1) *Preparation of animal and apparatus.*—For a study of the knee-jerk in an animal the nerve-muscle preparation isolated was the anterior crural-vasticrureus group. This was comparatively easy in the cat. The dissection as described by Sherrington [7] cuts all the muscles and nerves of the hip and thigh except the internal and external vasti and crureus muscles and the anterior crural nerve connecting them with the central nervous system. The proximal end of the peroneal nerve was also left for external stimulation. This dissection was usually bilateral, giving two knee-jerk preparations. In all cases both peroneal nerves were isolated. Thus we had an isolated reflex group, free from the influence of muscles acting on the same joint which might interfere with the recording of movement of that joint.

Cats were used for all experiments. The preliminary dissections described above to isolate the knee-jerk muscles were done under ether anæsthesia. The two peroneal nerves were exposed at the same time and glass-shielded electrodes attached to their proximal ends. A faradic current was used for stimulation of these nerves. The feet of the animals were amputated at the ankles and a pin attached firmly through the tendons and periosteum at the distal end of the tibiæ to provide a point of attachment for our recording apparatus. If a spinal preparation was to be used a preliminary laminectomy was done at

this stage, exposing the cord without opening the dura at about the tenth dorsal vertebral level. The skin was used to cover these operative defects so as to keep the limbs and back warm. All experiments were also performed on a warm table.

After the above steps, decerebration was carried out by using the Sherrington decerebrator. In practically every case decerebration was successful and good decerebrate rigidity was obtained. The animal was then placed on its back surrounded with hot-water bottles to insure a fairly even temperature. The head was slightly elevated. Animals could be kept in good condition for hours, in one case for over twenty-four hours. The legs were fixed by pivot pins through holes drilled in the condylar aspects of the femurs. A thread from the fixed pin at the ankle passed over a series of pulleys to recording levers, making a mark on a smoked kymographic drum. An upward fling of the leg (extension of the leg by contraction of the vasticrureus muscle), when the patellar tendon was tapped, recorded on the drum as an upward vertical mark. Variations in the tone of the muscle were recorded on the drum by the various levels of the marker. Thus a low level on the drum indicated low tonus (a low-tension muscle) [9], while a high level denoted a high tonus (high-tension muscle). Electrodes of the Sherrington [6] bipolar type were then attached to both peroneal nerves by separate circuits for stimulation by the faradic coil.

If a spinal preparation was desired the previously exposed spinal cord was cut entirely through at the tenth dorsal level, and the animal was then turned on its back and the same system of recording the movements of the vasticrureus as above described was used.

(2) *Variation of tonus in the isolated reflex group.*—Sherrington [8] had found that there was a marked increase in tonus after decerebration in extensor muscles such as the vasticrureus. This tonus of an antigravity muscle group can be decreased or increased by the stimulation of a sensory nerve of the same side or opposite side of the body. Thus, if the peroneal (partly sensory) nerve, which is convenient and accessible, be stimulated in the ipsilateral limb the tonus of the vasticrureus group is at once lowered. Vice versa, if the contralateral peroneal nerve be stimulated the tonus of this antigravity muscle group is increased. The relaxation of the extensor vasticrureus when an ipsilateral nerve is stimulated is part of the reciprocal innervation of the flexion reflex. Thus stimulation of ipsilateral and contralateral peripheral nerves enabled us to vary the tonus of this extensor muscle. By cutting the spinal cord in the mid-thoracic region we had a spinal

preparation with flaccid limbs and "toneless" muscles. Thus we could vary our tonus from complete rigidity, sometimes seen in the freshly decerebrate preparations, to flaccidity seen in the spinal animal, with variations between these two extremes by stimulation of the ipsilateral or contralateral peroneal nerves.

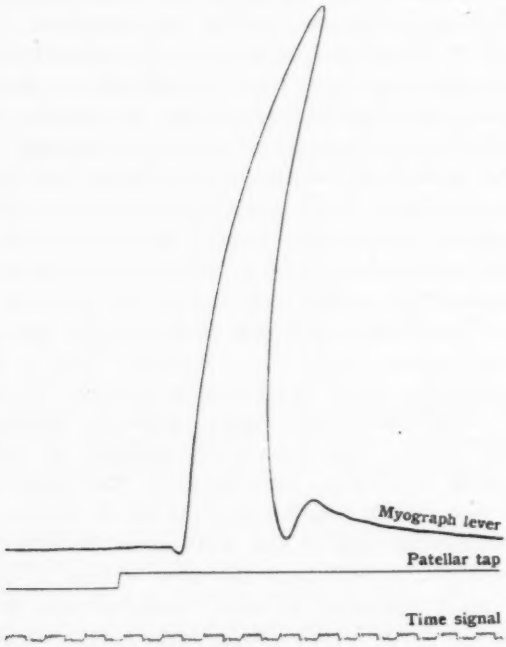

FIG. 1.—Myogram of the form of the isolated knee-jerk of a spinal cat. The upper line records the movement of the vasticrureus muscle when the patellar tendon is tapped. An upstroke indicates contraction of the muscle; a downstroke, relaxation. The approximate time of the blow of the patellar is indicated by the second line. The lower line is the record of a tuning fork vibrating 13·5 times per second.

III.—OBSERVATIONS.

(1) *The knee-jerk in the spinal and decerebrate cat.*—The response to a tap of the patellar tendon in a spinal preparation was first examined. The spinal cord had been cut through at the level of the tenth dorsal segment and the vasticrureus muscle showed very little tone. When the limb was elevated and let fall it would drop back to its original position. There was no "lengthening" or "shortening"

of the muscle on passive movement. After any manipulation the same level as before was recorded on the drum (i.e., the tension of the muscle remained constant). Although the muscle was flaccid to palpation the knee-jerk was fairly active. A unilateral record taken of this preparation is shown in fig. 1. The curve is a simple one of contraction followed by an almost parallel relaxation, the same level being maintained after the jerk as before. There was a slight rebound after the jerk. This rebound was thought not to be due to either mechanical bouncing of the knee-joint or the lever. It was presumed that the sudden elongation of the muscle after its contraction was enough of a stimulus itself to give another knee-jerk. With the cat on its back and the leg only supported by the distal end of the femur, the weight of the leg is, of course, a large factor in its return to its original position after the jerk. The second line of the record indicates the moment a hammer fell from its fixed position just above the patellar tendon. It does not, therefore, represent the exact moment the tendon was hit, for a slight amount of time was taken by the hammer falling a few centimetres. This record cannot be used, therefore, to measure the time of the reflex, as it gives approximately only the exact time of the stimulation.

Fig. 2 is a record of the same decerebrate animal without the spinal cord cut. In this experiment there was a moderate degree of decerebrate rigidity. All the extensor muscles of the upper limb were tonic and the vasticrureus was spastic to palpation. After the limb was extended from a flexed position by hand the muscle continued to stay shortened and showed a higher level on the drum than before it was moved. Vice versa, if the limb was flexed by hand a new level of tone was developed which continued without further change. The limb, therefore, showed the "shortening" and "lengthening" reaction and "plasticity," terms used by Sherrington to describe these phenomena; in short, the vasticrureus muscle could be "set" at various lengths and it would remain fixed at its new length. This plastic element in these spastic muscles was demonstrated in many decerebrate preparations, and we came to recognize it as one of the most constant signs in decerebrate rigidity. In this spastic vasticrureus muscle the knee-jerk showed a form quite different from the non-spastic or spinal muscle. Two elements would be easily recognized: the jerk which appeared first and resembled the jerk of the spinal knee-jerk, and the second factor, the plasticity. The jerk was slightly greater in amplitude than the corresponding jerk in the spinal animal. After the jerk, however,

the muscle did not completely relax but remained partly contracted at a shorter length than before it was stimulated. This shortening showed as a higher level on the recording drum. Thus there was a new tonic level after each knee-jerk more tonic than the level before the jerk. The muscle continued at this level or tonic state until another jerk or

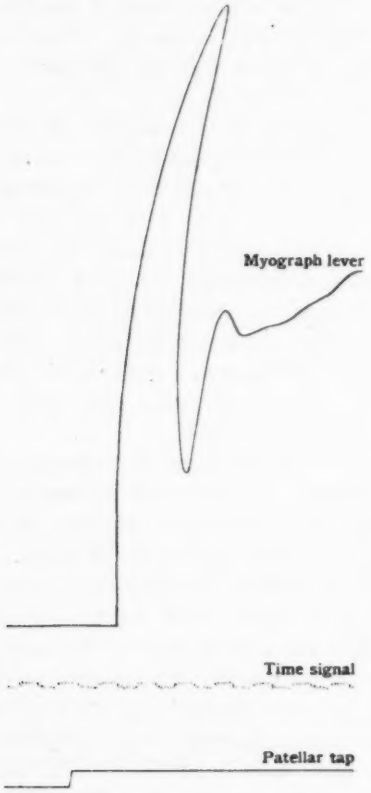

FIG. 2.—Myogram of the form of the isolated knee-jerk of a decerebrate cat. The tracings indicate movements as in fig. 1, except that the time record is placed above that of the patellar tap.

some other factor changed its tonus. The form of the knee-jerk, therefore, was changed in the decerebrate from the simple jerk seen in the spinal animal to a jerk plus a continued shortening of the muscle, the second element being the result of the "plastic tonus." Fig. 2 is a typical curve taken from many, and when compared with fig. 1 the

difference in the final phase of the knee-jerk is apparent. In the cat, then, we may say that increased tonus of the vasticrureus muscle (decerebrate) causes a change in the form of the knee-jerk, the jerk being slightly greater in amplitude, and also exhibiting plasticity not found in the muscles with slight tonus (spinal).

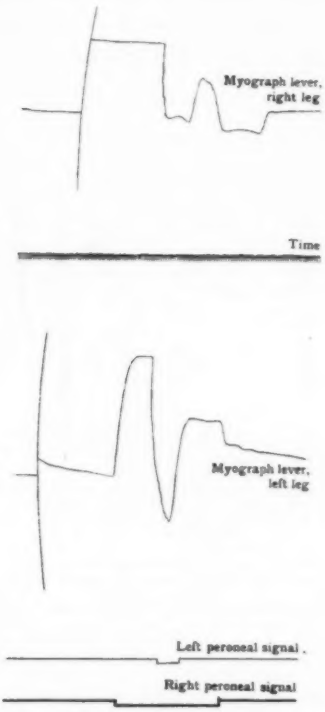

FIG. 3.—Reciprocal innervation in two isolated vasticrurei muscles. (See text for description.) An upstroke of the lever indicates increased tension of the muscles; a downstroke, decreased tension. Time, 13·5 beats per second.

We may vary the tonus of the vasticrureus muscle by stimulating the ipsilateral or contralateral peroneal nerves. Fig. 3 is a record of such an experiment. The movements of the two vasticrureus muscles are recorded as well as the time of the peroneal stimulations. The first nerve stimulated was the right peroneal. The tonus of the right extensor muscle was inhibited by this stimulation and the tension of the muscle relaxed, i.e., the writing point dropped; at the same time

the tone of the left vasticrureus muscle was increased, the tension increased and its lever was raised. While the tone of the right extensor muscle was lowered and that of the left raised, we stimulated the left peroneal nerve and the reverse of the above took place; the tonus in the right muscle was increased, and in the left muscle

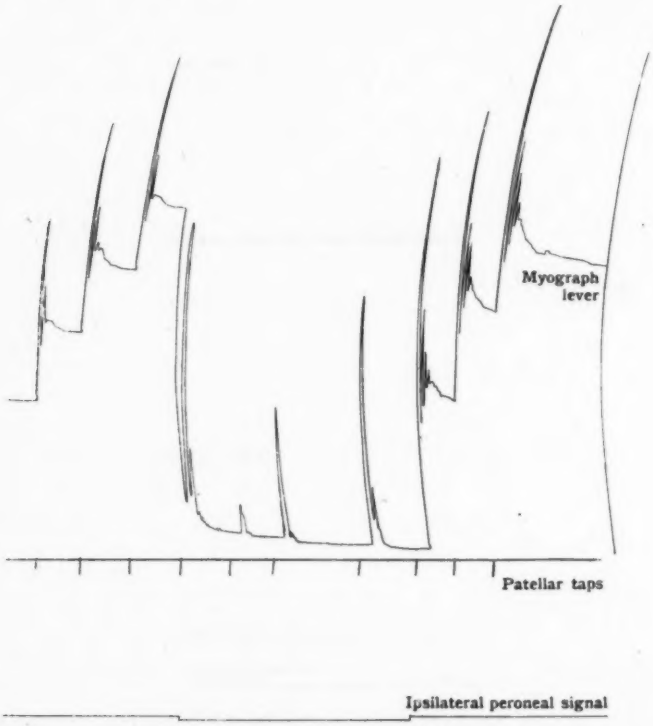

FIG. 4.—Showing the effect of ipsilateral peroneal stimulation on the form of the knee-jerk in a decerebrate cat. (See text for description.)

decreased. As the stimulation of these two peroneal nerves was removed by cutting off the faradic current in each, the original movements described above took place in the reverse order. It is clear, then, that we were able to raise or lower the tonus of these knee-jerk muscles by the use of contralateral or ipsilateral peripheral stimulation.

(2) *The knee-jerk in tonic variations.*—Fig. 4 illustrates an experi-

ment in a decerebrate preparation showing marked spasticity with the usual plastic element. The first three knee-jerks recorded show the increase of tonus after each jerk. This causes a "staircase" effect in the record, a condition characteristic of decerebrate rigidity in the extensor muscles of the cat. The tone of this extensor muscle was then lowered by stimulation of the ipsilateral peroneal nerve, and during this stimulation a number of knee-jerks were elicited corresponding to the spinal type of knee-jerk in each case. Although they varied somewhat in amplitude the relaxation after the jerk was as great as the

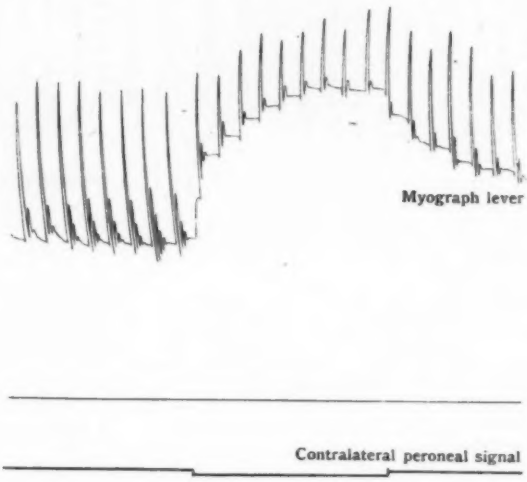

FIG. 5.—Showing the effect of contralateral peroneal stimulation on the form of the knee-jerk in a spinal cat. (See text for description.) Points of patellar taps not indicated.

contraction and the element of plasticity in the muscle has been eliminated. After the current was released the knee-jerk again assumed the form of the decerebrate preparation. Of the ten knee-jerks recorded in this figure the first three and the last three show the typical decerebrate form with plasticity, while the four middle ones show the simple twitch or spinal type. The middle line of the record marks approximately the time of stimulation of the patellar tendon, while the lower line indicates the beginning and ending of the ipsilateral peroneal stimulation. There was a slight tendency to clonus (pseudo-clonus) in all of the decerebrate knee-jerks, not so marked in the spinal ones.

As fig. 4 represents a spastic or decerebrate knee-jerk taking the

form of a flaccid or spinal knee-jerk by inhibition of the tonus, fig. 5 shows the reverse condition, a flaccid knee-jerk of a spinal animal becoming a spastic knee-jerk as in a decerebrate animal. This record

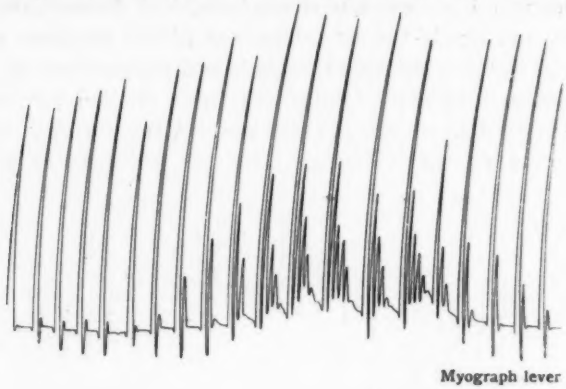

FIG. 6.—Slight increase in tonus with contralateral peroneal stimulation in a spinal cat with development of a pseudoclonus. Middle line indicates points of patellar taps. Lowest line shows point of peroneal stimulation.

is of an experiment on a spinal animal, and it can be seen that the knee-jerks at the beginning of the record are typical of the spinal type. Between the ninth and tenth knee-jerks on the record the contra-

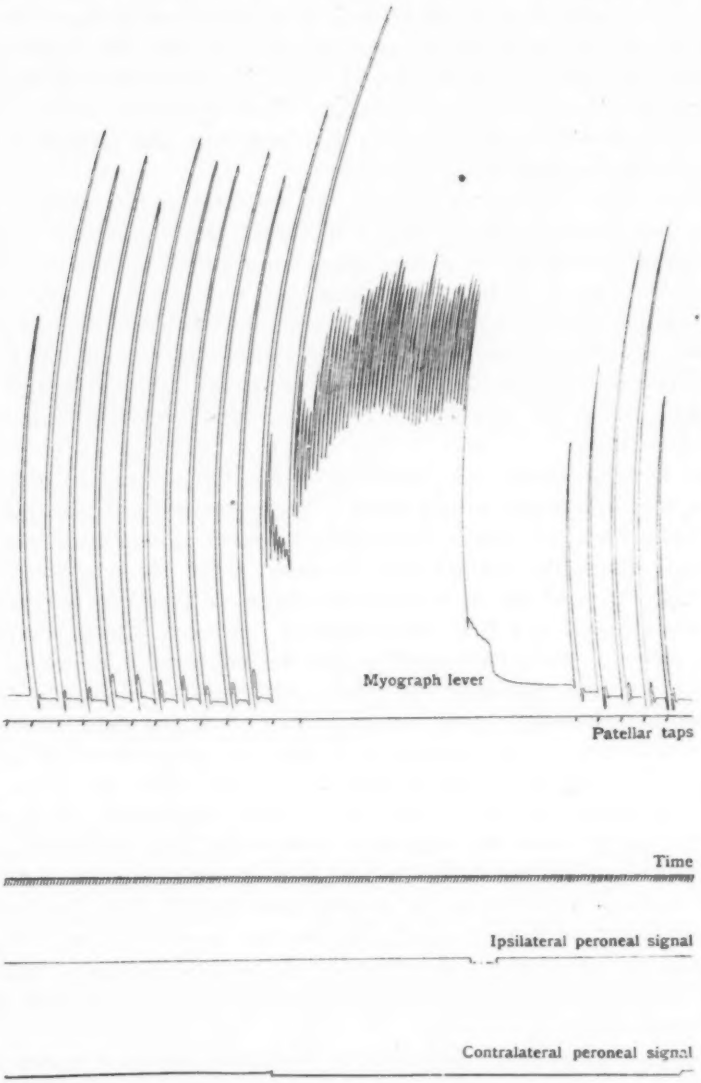

FIG. 7.—Knee-jerks in spinal cat augmented to a clonus by contralateral peroneal stimulation and stopped by ipsilateral inhibition. The first ten knee-jerks are "spinal" in form, At this point contralateral stimulation raised the tonus, and the next knee jerk was spastic. followed by another with clonus. The tonus was then strongly inhibited by ipsilateral stimulation, the clonus stopped and the knee jerks resumed their spinal form. The upper line is the myograph lever, below this, the signal of the patellar taps, the time 13·5 beats per second, ipsilateral peroneal signal, and below it, contralateral peroneal signal.

lateral, peroneal nerve was stimulated, thus increasing the tonus of the extensor muscle, and during this increase of tone the knee-jerks assumed the spastic form with spasticity. When the stimulation was released, the knee-jerks again resumed their spinal character. The lowest line of the record indicated the beginning and ending of the contralateral peroneal stimulation.

In the last two records, therefore, we have first a preparation with a high degree of tonus and plasticity showing the typical decerebrate knee-jerks reduced by ipsilateral stimulation to a preparation with a low degree of tonicity showing the spinal type of knee-jerk. On the other hand, the second experiment illustrates the exact reverse of this; a spinal animal with a typical spinal knee-jerk has its tonus in the extensor muscle increased by contralateral stimulation, and the knee-jerks show the decerebrate or spastic type while the muscle is thus stimulated.

(3) *Patellar clonus.*—A pseudoclonus was often noted in both the spinal and decerebrate preparations. There was more tendency for it to develop when the tonus was slightly elevated by stimulation of the proximal end of the contralateral peroneal nerve (fig. 6). It will be seen that the knee-jerk showed a slight degree of plasticity during this stimulation, and also that the "rebound" or second jerk became a short series of jerks from two to four in number. If, however, the tonus augmenting stimulus was slightly stronger than shown in fig. 6, a real self-sustaining clonus could be developed, the relaxation after each contraction being enough of a stimulus presumably to set off another jerk (fig. 7). During this experiment, when the tonus was suddenly dropped by an ipsilateral peroneal stimulation, the clonus at once stopped and the knee-jerk resumed its original spinal form.

Such a clonus was often started in a decerebrate animal with a spastic vasticrureus muscle by raising the tonus by the "staircase" method of repeated knee-jerks. Fig. 8 is the record of an experiment showing that one patellar tap was sufficient to cause a pseudoclonus and raise the tonus enough so that the second tap was followed by a continued or actual clonus.

Fig. 9 illustrates an experiment in which the clonus is shown in a little more detail. This tracing, from an experiment on a decerebrate cat with a spastic vasticrureus muscle, illustrates the development of a clonus after a single knee-jerk raised the tonus to a certain level. It also illustrates the regularity of the contraction and relaxation of the muscle during the clonus. The rate of the clonus in

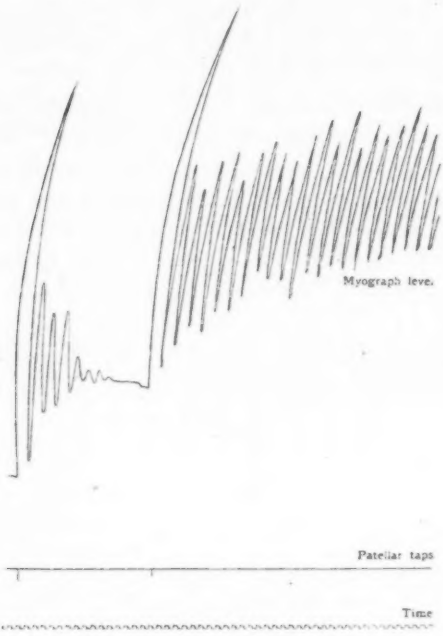

FIG. 8. - Clonus in vasticrureus muscle of decerebrate cat inaugurated by successive knee-jerks, the "staircase" effect. The middle line is the signal for patellar taps. The lowest line is the time record, 13·5 beats per second.

FIG. 9.—Patellar clonus in decerebrate cat, vasticrureus muscle, inaugurated by single knee-jerk. Rate of clonus, 11·5 jerks per second. Time, below, 13·5 beats per second.

this experiment was 11·5 complete contractions per second. Other records of patellar clonus were 11·2, 12·6 and 11·5 complete contractions per second. In still other animals the rate was higher, 17·2, 17·1, 17·3 and 16·4 complete contractions per second. There seemed to be, therefore, two rather definite rates, one about twelve complete beats and

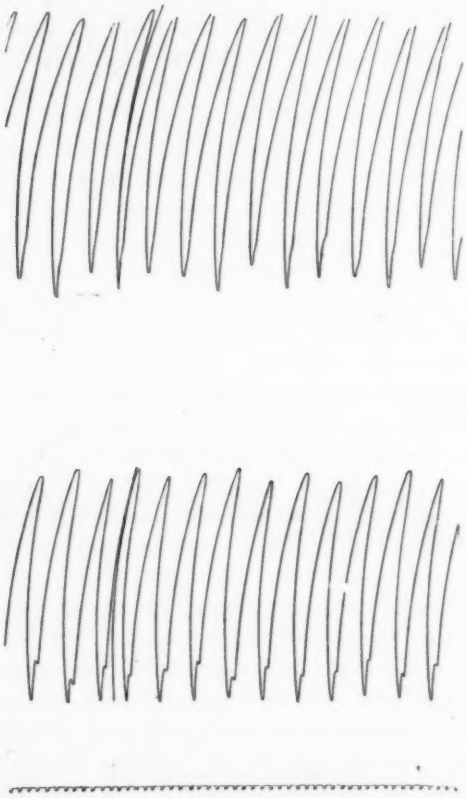

FIG. 10.—Reflex "walking" in a decerebrate cat; two vasticrurei muscle preparations. Alternate contraction of the two muscles. Rate, 3·5 steps per second. (For full description, see text.)

another about seventeen complete beats per second. The rate was always nearly constant in the same animal.

One case of patellar clonus was studied in man, using the same apparatus. The case was one of a hemiplegia of some years' duration with marked spasticity of the left leg, increased knee-jerks, patellar

clonus, ankle clonus and a positive Babinski reflex. The rate of the patellar clonus in man was between 8·8 and 9·7 complete beats per second.

By way of comparison with the clonus rate some records were obtained of the "walking reflex," an alternate contraction and relaxation of the vasticrurei, and fig. 10 shows a record of the two extensor muscles during the walking reflex. The rate of this walking at the time this record was taken was 3·5 steps per second, but great variations in the rate were observed. At one time it was recorded that the rate was 1·7 steps per second and at another 2·7. It will be seen, therefore,

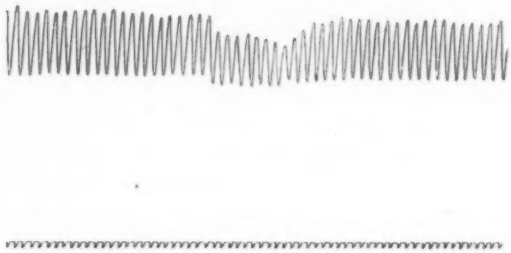

FIG. 11.—Patellar clonus in a decerebrate cat; vasticrureus muscle. Tonus inhibited by ipsilateral peroneal stimulation, indicated by signal, lowest line. Time, middle line, 13·5 beats per second.

that the walking reflex is quite different from the patellar clonus. The patellar clonus is very constant in the same animal, although it may be of different rates in different animals. The walking reflex, on the other hand, varies in rate from time to time in the same animal.

A double clonus was never successfully recorded in any of the experiments, although it was observed a number of times. The contractions were synchronous, not alternating as in walking. Clonus was not always obtained in decerebrate cats, but appeared at unsuspected times, often when our apparatus was not adjusted for recording owing to mechanical difficulties. We were never able to obtain clonus by any special form of decerebration and it was always observed incidentally while working on the form of the knee-jerk.

Variations in the form of the clonus were observed during changes in the amount of clonus exactly corresponding to variations in the form of the knee-jerk during changes of the tonus. Fig. 11 is a tracing of an experiment in which the tonus was decreased by an ipsilateral peroneal stimulation during the recording of a clonus. The record shows the drop in tone and also that the clonus changed slightly in character. The rate remained the same, but the clonus became irregular with variations in the size of the jerk. When the inhibition of the tonus was released the clonus gradually " climbed " and sought the same

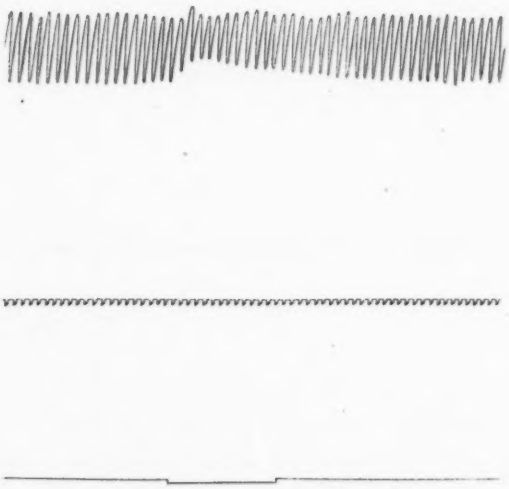

FIG. 12.—Patellar clonus in a decerebrate cat; vasticrureus muscle. Tonus augmented by a weak contralateral peroneal stimulation, indicated by signal, lowest line. Time, middle line, 13·5 beats per second.

level (i.e., tone) as before inhibition. On the other hand, when the tone was increased by contralateral peroneal stimulation the same phenomena were observed (fig. 12). There was a decrease in the amplitude with a slight irregularity. If the tonus was decreased or increased markedly the clonus would at once stop and it was observed that only between certain degrees of tonus (*i.e.*, certain levels on the drum) could clonus be obtained, and any tonus above this level or below this level was sufficient to stop the clonus.

IV.—Discussion.

There are two distinct forms of the knee-jerk noted in these experiments. One is the simple jerk with the contraction and relaxation of about the same degree, as seen in the spinal animals. The other is the decerebrate knee-jerk in which in addition to the twitch there is a plastic element which holds the muscle at a new degree of tonic level after the jerk. This second element in the decerebrate knee-jerk was shown by Sherrington [7] in his experiments on the shortening and lengthening reactions. He showed that the spastic muscle in the decerebrate cat was capable of being "set" at various lengths and would remain so for an indefinite period. These two elements in spastic muscles in decerebrate animals have been noted by other investigators. Langelaan [3] proposed the terms "contractile tonus" and "spastic or autonomic tonus," and clearly showed them in his experiments on animals. He concludes "that the contraction following the tap on the tendon is of a twofold nature, viz., a twitch upon which is superposed a tonic contraction."

Plasticity as a part of spasticity has not, so far as I am aware, been adequately demonstrated in man except for Wertheim Salomonson's paper. He obtained, in a case of cerebral diplegia, curves of the knee-jerk exactly corresponding to the spastic curves seen in the cat (fig. 2.) He noted that "the muscle does not entirely relax, but a state of hardening of the muscle substances persists for a longer or shorter time." From the illustrations he uses, the "hardening," I think, is the same as the plastic tonus described by Langelaan and others. The twitch is followed by a continued contraction as in the experiments on animals. I have, however, noted plasticity in some of the severe cases of hemiplegia and in one case of diplegia, but have never recorded this graphically. The so-called "lead pipe" rigidity is, I think, an example of plasticity combined with spasticity in man. Nevertheless it is certainly not the usual finding in a spastic muscle in man, as the most noticeable phenomenon is the great increase in the amplitude of the responses to the patellar tap. Thus the laboratory finding, where plasticity is the usual companion of spasticity, is not always easily demonstrated in the clinic.

In the laboratory, however, we are observing animals whose nervous system is far less integrated than the nervous system of man. The activity of the spinal animal where reflexes may be demonstrated in almost pure form is much greater than the "spinal man." It must be recognized, nevertheless, that many of the reflexes such as the scratch

reflex and the extensor thrust, so clearly demonstrated by Sherrington in animals, are present in man, although they may be so weak or changed that they are not easily found. The work of Head and Riddoch [2] on certain reflexes in cases of complete spinal cord lesions without infections shows that many more reflexes may be seen in the isolated human spinal cord than was previously supposed. Riddoch [5] found that the usual knee-jerk in his cases of complete section of the spinal cord was a simple twitch, presumably the same as is shown to be the case in spinal animals (fig. 1). In one of his cases, however, where there was not a complete transection of the cord he found after the twitch of the knee-jerk "the leg fell away more slowly and the relaxation of the tendon was more deliberate." He thought in this case there was some "plastic tonus," as well as "contractile tonus," to use Langelaan's term. Walshe [10] has also successfully searched in men for reflex actions that Sherrington had demonstrated in the laboratory.

It is, perhaps, not remarkable that the plastic element in spasticity has not been observed more often in man. It must be remembered that one seldom has a chance of seeing in man an isolated reflex group such as is artificially produced in animals. Also, the extraneous factors which I mentioned in the early part of this paper are so profound that one cannot observe a pure reflex contraction without the action of other muscles on the same joint or inhibition or augmentation of the reflex by voluntary control. It was observed in these experiments that it was not possible to obtain a knee-jerk in an absolutely toneless muscle. In the spinal animal the vasticrureus muscle was not entirely toneless, for we were able to inhibit some tonus by ipsilateral peroneal stimulation. In other words, we were able to decrease its tonus and finally a point was reached where no reflex was obtained. This, I think, demonstrates that tonus is not entirely dependent on control from centres in the mid-brain, for when the spinal cord is entirely cut across we have severed all connection with such centres. Such a spinal animal has very little tone and we speak of the muscles as flaccid, but nevertheless there is some tonus left. This point, too, has been demonstrated in man by reflexes obtained after complete transverse lesions of the spinal cord. Such reflexes I believe are not possible without some tonus.

It was noticed that the spastic vasticrureus muscle when "set," well lengthened, and then stimulated by blows on the patellar tendon, tended to shorten at each jerk until a certain level was reached on the recording

drum, and then tended to remain at that level. In other words, the muscle found a length between its high and low tonus length, at which point there was a marked tendency to remain. This level was also shown in the experiments where the tonus was decreased or increased by ipsilateral or contralateral peroneal stimulation; after such stimulation the muscle took what I have called a "neutral tension." Two factors seem to control this tension, one the reflex tonus tending to increase the tension of the muscle and the other the weight of the limb tending to pull it down and thus lengthen the muscle. When the reflex tonus was very great the muscle was shortened as far as the knee-jerk would allow, the limb being completely extended and the weight of the limb being completely overcome. The point of interest is that at a certain level of tonus, clonus was most apt to appear. It is at the neutral tension of the muscle somewhere between its high and low tonic level that clonus is most apt to appear. In other words a muscle at this tonic tension is in its best position to maintain a continued series of contractions. Clonus was obtained mainly in the spastic muscles at their neutral tension. If a spastic vasticrureus was set at a low level of tonus and the patellar tendon tapped, the plasticity after the jerk brought the muscle to the most favourable level before clonus would appear. If the tonus during a clonus was increased or decreased by contralateral or ipsilateral peroneal stimulation and passed beyond the narrow limits of its neutral tension the clonus either became irregular or disappeared. Langelaan has noted that "a clonus is composed of a series of twitches superposed upon a tonic shortening." This, I think, is correct in a measure, but the tonic shortening (or increased tension) must be a definite one, for any increase or decrease in the tension of the muscle beyond the narrow limit of its neutral tension is enough to cause the clonus to stop. Wertheim Salomonson found clonic after-contractions (pseudoclonus) in many of his cases and in others stated that "the tonus is not quite high enough to permit of a clonus." He showed no records of a true clonus.

It would seem then that a true clonus is a series of continuous reflex twitches dependent upon a more or less fixed tone of the muscle. Clonus is possible only over a slight range of tonus, for there is a tonic tension of a muscle, a "neutral tension," that is, the tone "of choice," for a series of continued self-sustaining twitches. If the plastic element holds the muscle at this length, then the contractile force will produce the jerk, and a clonus will result.

The clonus, as we have noted, is a regular series of twitches. If the

tone is raised or lowered, the clonus becomes irregular in both amplitude and rate. Thus we have in our plastic tonus the mechanism by which such a continued muscular effort as the clonus is controlled, so that it continues regularly and smoothly. The tonic level is so regulated by plasticity that the muscle is set at a correct height, so that the falling limb is just enough stimulus to provoke the next twitch. This seems to me one of the functions of plasticity, and its value in animal economy cannot be doubted.

Conclusions.

Experiments on cats, both spinal and decerebrate, with the anterior crural-vasticrureus nerve-muscle preparation isolated in situ, and the movement of the knee-joint recorded graphically, gave the following results:—

(1) In the records from the spinal animal the knee-jerk is a single twitch, the contraction and relaxation of the extensor muscle group being almost parallel. After the jerk there may be another jerk ("rebound") or a short series of jerks ("pseudoclonus"), but the length of the muscle after each jerk is the same as before the experiment.

(2) In the records of the decerebrate animal the knee-jerk is a single twitch followed by a continued contraction, the muscle assuming a new and shorter length after the jerk. Thus, there are two elements in the phenomenon, the twitch or jerk and the continued contraction. The first may be termed the "contractile element," and the second the "plastic element," after Langelaan.

(3) The plasticity is an indicator of muscle tonus. When the tonus is varied by artificial means, the amount of plasticity varies also. When the tonus is lowered by an ipsilateral peripheral nerve stimulation, the plastic element may drop out of the knee-jerk record, and the jerk takes the form seen in the spinal animal.

(4) Some tonus can be demonstrated in the flaccid vasticrureus muscle of the spinal animal, for it may be reduced by ipsilateral peripheral nerve stimulation, showing that the distal portion of the completely severed spinal cord in a cat is capable of maintaining some tonus in the muscles of the lower limbs.

(5) Many of the knee-jerks in both spinal and decerebrate animals may show pseudoclonus; but it is most often found in decerebrate preparations, showing little tonus.

(6) A true clonus may be obtained in various ways. If the tonus of a spinal animal is raised by contralateral peripheral nerve stimula-

tion, a clonus may result. Decerebrate animals with moderate tonus most often exhibit clonus. The degree of tonus of the muscle at the time of clonus is termed its "neutral tension." This tension is a neutral point between the reflex tonus, tending to increase the tension of the muscle and weight of the limb tending to lengthen it. This tension, and this tension only, is the most favourable point for the maintenance of a clonus.

(7) A true clonus is characterized by a continued series of self-sustained reflex twitches, regular in rate and amplitude, markedly altered or stopped by increasing or decreasing the tonus.

(8) The rate of a clonus in the vasticrureus muscle group of a cat was found to vary, most of the records falling into two groups—a rate about 12 per second and another about 17 per second. Patellar clonus in man was found to be 8·8 to 9·7 per second.

(9) The rate of "walking" varied from 1·75 to 3·5 steps per second.

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ALTERATIONS OF THE GOLGI APPARATUS IN NERVE CELLS.[1]

BY WILDER G. PENFIELD, M.A., M.D.

Retispersion.

An intracellular reticulum was first described by Golgi [10] in 1898, in the cells of the central nervous system. This observation has been greatly extended, first by his pupils in Pavia, and subsequently by a large field of workers, until at the present time the structure has been described in the cytoplasm of practically all types of cell throughout the animal kingdom [24, 6, 7, 21]. The structure has received a variety of names, such as the internal reticular apparatus of Golgi [10], the "Binnennetz" of Kopsch [15], the canalicular apparatus (Bensley [1]), &c.

This study is concerned only with the apparatus as seen in nerve cells, where it appears in its most highly differentiated form and presents normally great variation in appearance. There have been numerous theories as to its function in the cell. But before a study of this problem can be logically undertaken, an inquiry must be made into the alterations of this structure in varying nerve cell conditions. Such an inquiry is the purpose of this paper.

The Golgi apparatus presents normally, in the great majority of cases, a complete attenuated reticulum with many varicosities or lacunæ (figs. 1 and 2). These varicosities may be homogeneous and black, as though full of some substance in which the silver is deposited, or they may frequently appear empty of such substance, and only the surface or boundary of the space take on the silver. The structure is confined to the cytoplasm, never encroaching on the nucleus or the periphery of the cell. Threadlike prolongations may pass outward into the dendrites, but I have never seen them in the axone process, nor have I found any such description in the literature.

Variations from the complete reticulum are most frequently seen in

[1] This research forms part of the work done for the degree of B.Sc., in the Physiological Laboratory, Oxford University. Expenses were defrayed partially by the Christopher Welsh Scholarship Fund.

the spinal ganglion cells. The whole structure appears rarely in one half of the cytoplasm only. It may also, occasionally, be fragmented to a greater or less degree. It may be hypertrophied or meagre, but under normal conditions the general pattern is surprisingly constant for each type of nerve cell.

A Zeiss objective, homog. immers. 1·5 mm., and a 6 compensating eye-piece (magnification 1,000) were used for all drawings, which were made with the aid of an Abbé camera lucida. Zeiss. All sections were prepared by the Cajal uranium-formol-silver method without counterstain. All material was obtained from cats.

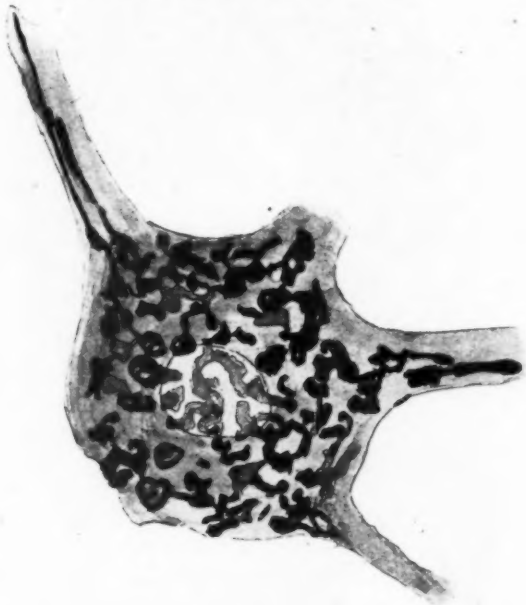

FIG. 1.—Normal cell in anterior horn of spinal cord. That part of the Golgi apparatus which is grey instead of black was drawn with the focus at a deeper level, below the nucleus.

THE LITERATURE OF ALTERATIONS IN THE GOLGI APPARATUS.

We have to thank the brilliant researches of S. Ramón y Cajal and his pupils for the larger part of our knowledge of this subject.

Autolysis.—Autolytic changes in the Golgi reticulum are rapid, beginning in man, according to Cajal, two hours after death. At twenty-four hours the reticulum has become granular, and in some cells only fine fragments will be found to remain.

In the cells of transplanted ganglia, the apparatus shows similar changes [23, 24].

Trauma.—In 1914, when studying the changes following traumatic inflammation, Cajal [23] concluded that the reticulum is more resistant

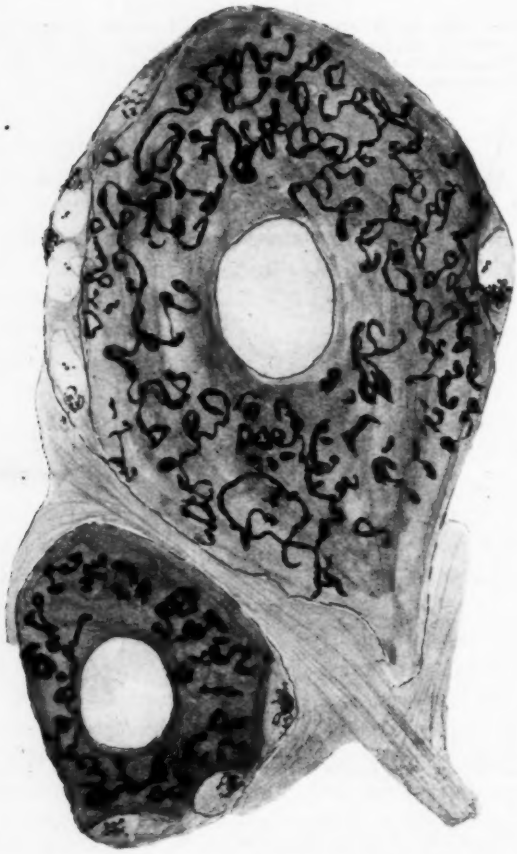

FIG. 2.—Normal cells in spinal ganglion. About both nerve cells may be seen the subcapsular cells, whose Golgi apparatuses are located at one pole of the respective nuclei.

to pathological agents than are the neurofibrils. But he found in the nerve cells of the cord and the pyramidal cells of the cortex that direct trauma causes fragmentation of the reticulum and its displace-

ment to the cell periphery and into the bases of the dendrites. The cell undergoes a process similar to chromatolysis, and as the nucleus approaches the periphery fragments of the Golgi apparatus frequently gather near it. The reticulum may disappear altogether. Therefore, in chromatolysis subsequent to direct injury, the Golgi apparatus and Nissl granulations behave somewhat similarly, both moving out to the periphery of the swollen cell and sometimes disappearing. In later stages, six to twelve days after the wound, he found on its border nerve cells with frankly hypertrophied Golgi apparatus.

Faradism.—Legendre [16] stimulated the posterior root of a lumbar spinal ganglion. He found that, as a result, the Golgi net in each cell was displaced to the periphery and became somewhat deformed. It is not stated what precautions were used to prevent spread of the electrical current along the root to the ganglion direct. Holmgren [12, 13] also tried the effect of electrical stimulation on nerve cells and found that, accompanying chromatolysis of the Nissl bodies, there resulted (*a*) "Kanalizirung," an emptying of the canals, and (*b*) diminution in size of the trophospongium, a system of canals having extracellular connections, which he maintains [14] is identical with the Golgi apparatus.[1]

Lesion of Axone.—Marcora [17, 18] found that, subsequent to section or tearing out of the hypoglossal nerve in a rabbit, there developed a fragmentation and displacement of the intracellular apparatus toward the periphery, which was well marked on the fourth day and reached its height between the fifteenth and eighteenth days. He also noted hypertrophy of the Golgi apparatus after the immediate effect of section had passed.

Cajal [24] described lobulation and fragmentation of the motor cells of the spinal cord after section of the corresponding nerves. He stated that this change accompanied chromatolysis, and, as the nucleus approached the cell periphery, the remains of the broken reticulum often gathered about it, or in the bases of the dendrites. He found no change in the Golgi apparatus of the cells of Clarke's column after section of their axones and, similarly, after section of the sciatic, he reported the spinal ganglia of that side unchanged. He concluded that section of the peripheral extension of a sensory neurone is not a

[1] The question of the identity of these two structures is discussed in a paper to appear shortly [22]. For an excellent summary of the literature on this subject, see Duesberg [6, 7].

sufficient cause to produce noticeable changes in the Golgi apparatus of that cell.[1]

It is of interest that Cajal [25] showed changes of the internal reticulum in the cells of the sheath of Schwann on the peripheral stump of the nerve after section. The reticulum became increased in quantity, occupied almost the whole width of the sheath, and extended along the fibre in either direction from the nucleus in parallel lines.

Nissl Granulations—Chromatolysis.

Inasmuch as many of the changes reported below in the Golgi apparatus are associated with somewhat similar changes in the Nissl granulations (chromaphil substance), it may be of interest to review briefly our knowledge of the latter and the inter-relation of the two substances.

The most delicate morphological index of the functional activity of a nerve cell is generally considered to be the appearance of the chromaphil substance in the cytoplasm, the so-called Nissl granulations.

Alterations in the appearance of this substance caused by electrical stimulation (Nissl, Vas, Lambert, Mann, Lugaro, Pugnat, and others) consist in a progressive diminution associated, in some cases, with displacement of the granules toward the periphery of the cell. Faradic stimulation creates abnormal conditions, however, which are probably not reproduced in the physiological activity of the nerve cell.

The results obtained by a long series of investigators (Van Gehuchten [30], p. 300) indicate that, following normal cell activity, there is a diminution in the amount of Nissl substance, which grows more marked with the onset of fatigue.

After section of the nerve, there may result, as first pointed out by Nissl [20], a change in the cells, the most prominent features of which are: disappearance of the Nissl substance, a considerable increase in the size of the cell, and displacement of the nucleus toward the cell periphery. This phenomenon was called by Marinesco chromatolysis, inasmuch as the alteration in the Nissl substance seemed to be its most striking characteristic. The first signs of change may appear as early as forty hours after the section. (Van Gehuchten [30], p. 305.) The Nissl

[1] Page 69 of reprint from Reference 24. "Debemos, pues, concluir, que la sección de la rama periférica de la neurona sensitiva no es causa suficiente para provocar perturbaciones marcadas en el aparato de Golgi de esta célula."

granules begin to disappear at the centre of the cell, and are visible last about the periphery and sometimes close to the nucleus. Following this phase of "dissolution," which reaches its height after fifteen to twenty days, there ensues a phase of "reformation" which progresses rather more slowly until the cell is again normal in appearance. A certain proportion of cells never recover, but go on to atrophy. The duration and degree of chromatolysis depend on the type of injury to the axone. If the nerve be torn out, the reaction is more severe and certain than that which follows section, ligature, or pressure of the nerve.

In this paper we are most interested in the cells of the spinal cord and ganglia. The motor cells of the anterior horns of the cord appear to offer considerable resistance to section of the axone. At times there is no resultant chromatolysis (Van Gehuchten [29]). After a lesion of the peripheral neurone-outgrowth of a spinal ganglion cell, chromatolysis is rapid, but section of the central fibres gives rise to no evident reaction. Section of the spinal cord produces in the cells of Clarke's column, located below, an energetic and typical response which is eventually followed by complete atrophy of at least the large cells in the column. (Sherrington and Laslett [28].)

The Golgi Apparatus and Nissl Granulations.

In 1910 Legendre [16] proposed that these two structures were identical. He called attention to the fact that, in general, their distribution was the same, both being situated in the cytoplasm, separated from the cell periphery by a clear space and avoiding the axone hillock. When the Nissl granulations were small in size, the Golgi net was composed of small granules and fine filaments. After electrical stimulation of the nerve, he noted similar alterations of the two structures. In transplanted ganglia, he observed the disappearance of the net and granulations at about the same rate.

This close association of the two structures is of interest, but that Legendre was mistaken as to their identity has been amply proven by the observations of other workers. Collin and Lucien [4] impregnated the Golgi apparatus and stained the Nissl granulations in the same sections. They found a certain number of cells where the one was perinuclear and the other peripheral. Marcora [17] stained the two structures simultaneously and demonstrated the Nissl bodies lodged in the mesh of the Golgi net; and in embryonic tissue [19] he showed that the two were separated by a clear unstained space. Cowdry [5] demonstrated the independence of Nissl bodies, "canalicular apparatus,"

and also mitochondria and neurofibrils in the same cell, by the Bensley method.

Cajal [24] agrees to the similarity of distribution of the two protoplasmic structures with regard to the peripheral freedom of the cell from both and the resemblance of a fragmented reticulum to the Nissl granules. But he calls attention to the fact that, whereas the Nissl bodies are found in the nerve cells only, the reticulum of Golgi is found generally in cells of all descriptions. He reports that his preparations, uranium nitrate-silver impregnation followed by staining of the chromophil bodies with aniline dyes, show constantly a clear space surrounding the Golgi reticulum and separating it from the Nissl substance. He states that this clear space is certainly occupied by neurofibrils.

Technique.

In the following experiments the Golgi apparatus was demonstrated by Cajal's uranium-formol-silver method [24],[1] with several minor modifications, as follows:—

The tissue was removed from the body immediately after death. Blocks of the cord must be very thin, not over 1 to 2 mm. After an hour in the fixative, blocks may be removed and cut down more easily to the desired thickness.

(1) Fixation for twelve hours. Shake from time to time. Uranium nitrate, 1 grm.; commercial formol (40 per cent.), 20 c.c.; distilled water, 80 c.c. The formol should be neutral. It is sufficient to keep chalk in the bottom of the container. For the cord and spinal ganglia 20 c.c. of formol gives more consistent results than 15 c.c. as suggested by Cajal. It is best to make up solutions of formol and of uranium nitrate separately, of twice the required strength, and mix in equal parts, just before use.

(2) Impregnation for forty-eight hours. After washing in distilled water for a few seconds only, immerse in 1·5 per cent. silver nitrate. Shake from time to time.

(3) Reduction for twelve hours. (First wash in distilled water.) Hydroquinone, 2 grm.; formol (commercial), 20 c.c.; sodium sulphite, 0·5 to 1·5 grm.; distilled water, 80 c.c. Sufficient sodium sulphite should be added to turn the solution quickly to a coffee colour.

(4) Dehydrate very rapidly in alcohol. Pass through xylol to wax.

[1] An excellent summary of this method has been published in English by Carleton [2].

From 90 per cent. alcohol until the tissue is embedded should not occupy more than five hours. If this stage is not carried out rapidly it may be found that all silver will have disappeared from a peripheral zone of cells in the spinal ganglion sections.

The fixation is apparently not sufficient to prevent the subsequent solution of some or all of the constituents of the Golgi apparatus in strong alcohol (Gatenby [9]), if exposed to it for too long a time. Toning of the sections with gold chloride adds nothing.

In two experiments I obtained the best results and perfect fixation of the cord by subarachnoid injection of the fixative before death, as follows: The animal is anæsthetized and a laminectomy performed in the lower lumbar region, exposing the membranes. A medium-sized lumbar puncture needle is then passed into the subarachnoid space a short way upward beside the cord and the fixative allowed to flow in under a gravity pressure of 75 cm. The heart stops after about a minute of the injection, which is continued for twenty hours. At the end of this time the cord is removed, blocks cut and dropped directly into the silver nitrate bath. Likewise, sections from tissue which has been fixed in this way and left a few days in 95 per cent. alcohol give the most perfect result when stained by Nissl's method. Subarachnoid injection does not fix the spinal ganglia properly, if at all.

This method, in general, if adapted to the type of cells one wishes to study, gives very complete and fairly consistent impregnations of the Golgi apparatus. In my experience, strength of fixative and period of fixation are the most important elements in the adjustment of the method to any particular tissue. In the sections prepared for this study there was also impregnation, at times, of the mitochondria in the nerve cells, and the Golgi apparatus in both the neuroglia cells of the cord and the intracapsular cells (Holmgren's "trophocytes") of the spinal ganglia. In the ganglion cells an intra-nucleolar body (*see* Carleton, [3]) was frequently impregnated.

When counterstain was desired, Unna's polychrome methylene blue was employed, demonstrating Nissl granulations, nuclear structures, &c.

For the purpose of this research, cats were used, under two to three months of age as a rule. In the few experiments where older cats were used, the impregnation of the Golgi apparatus (contrary to the experience of Cajal) was equally satisfactory. The frequent presence of well-stained pigment granules, however, in the material from the older animals, added another cytoplasmic constituent.

Results of Experiments.

To reconsider briefly some of the foregoing paragraphs: As a result of section of the axone, changes in the Golgi apparatus have been described in the motor cells of the cord and bulb, but none in the cells of the spinal ganglia or Clarke's column. The nature of the reported change is progressive fragmentation and lobulation of the reticulum with some peripheral displacement. Chromatolysis appears in the cells of the anterior horn and Clarke's column after axone section, and

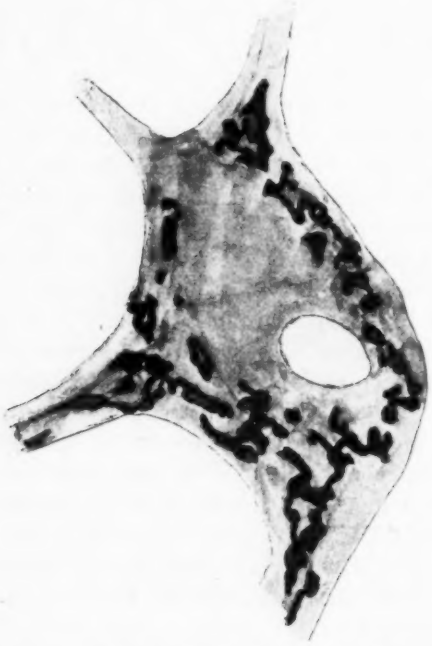

Fig. 3.—Retispersion in anterior horn cell of left side of spinal cord. Level of 7th lumbar segment. Seven days after section of left sciatic nerve.

in spinal ganglion cells after section of the peripheral extension of the axone only. Cutting of the central extension fails to cause the reaction. My observations give no cause to modify these conclusions, at least in so far as the phenomenon of chromatolysis is concerned.

Positive results.—The work reported here indicates that the Golgi apparatus in all types of neurones studied, afferent as well as efferent, responds to an axone lesion in a consistently specific manner.

The *motor cells* of the cord were studied after simple section of the sciatic nerve. There was no possibility of trauma as the section was made at the level of the great trochanter, a distance of about 5 cm. below the seventh lumbar ganglion when measured along the nerve. At the end of seven days, after the cutting of the nerve on one side, the reticulum had begun to leave a clear zone about the nucleus of the motor cells on that side. This could be made out in 22 per cent. of the cells counted. No such condition was found in the examination of an equal number of cord neurones on the opposite side, where the nerve was intact. Aside from this peripheral displacement, there was very little change in the appearance of the apparatus (fig. 3). After sixteen days the change had become more general, the clear perinuclear zone was wider and now appeared in 50 per cent. of the homolateral motor cells, the heterolateral side being 100 per cent. normal. There was now a moderate amount of fragmentation in some cells, while in others the reticular structure remained unbroken. The neighbourhood of the axone base becomes free of all Gogli apparatus early in the process.

It seems desirable to call this process by a distinctive name, *retispersion* (from *rete*, a net, and *spargo*, to scatter), especially since it may appear independently of chromatolysis as will be shown below. After this stage of dispersion of the Golgi apparatus to the cell periphery, there may succeed a stage of dissolution, when the reticulum becomes progressively fragmented. Thus *retispersion* may be followed by *retisolution*. It seems evident, however, that the amount of retisolution varies with the different types of neurone.

The cells of *Clarke's column* respond in a most energetic fashion. In Experiment 14, the spinal cord of the cat had been completely severed four days previously. The section was at the level of the eleventh dorsal spine. In the lower lumbar region, the cells which correspond to Clarke's column higher in the cord showed a striking change. The Golgi reticulum was close against the periphery of the cell except where some remained about the nucleus, which also had become eccentric. In spite of its extreme dislocation, however, the network of the apparatus was complete for the most part (fig. 5). Retisolution had begun only in a few cells (fig. 6). Nissl granulations were absent.

The *spinal ganglion* cells present normally such a rich variety of types of Golgi apparatus that considerable care must be taken to control all conclusions. Four days after section of the left sciatic, retispersion had begun in the spinal ganglion cells of the same side. The Golgi apparatus had moved outward toward the periphery, leaving a clear

space about the nucleus in 48 per cent. of these cells, whereas in the same ganglion on the opposite side the apparatus was more or less peripheral in only 16 per cent., which variation is about the average for

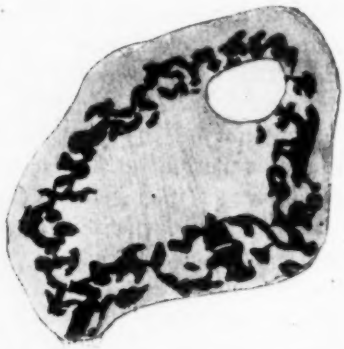

FIG. 5.—Retispersion in neurone of Clarke's column in lumbar spinal cord. Four days after section of spinal cord at level of 11th thoracic spine.

FIG. 6.—Same as fig. 5, showing considerable retisolution as well.

normal ganglion cells. The difference,[1] of course, is more especially one of degree of displacement, which cannot be estimated in figures. Nissl stains showed no evidence of chromatolysis as yet.

[1] If sections are cut thin, at about 6 μ, retispersion is less apt to be overlooked than when the sections are thick.

Seven days after the sciatic was cut, the peripheral dislocation of the reticulum reached its height, affecting 80 per cent. of the homolateral cells as compared with a count which was again 16 per cent. for the

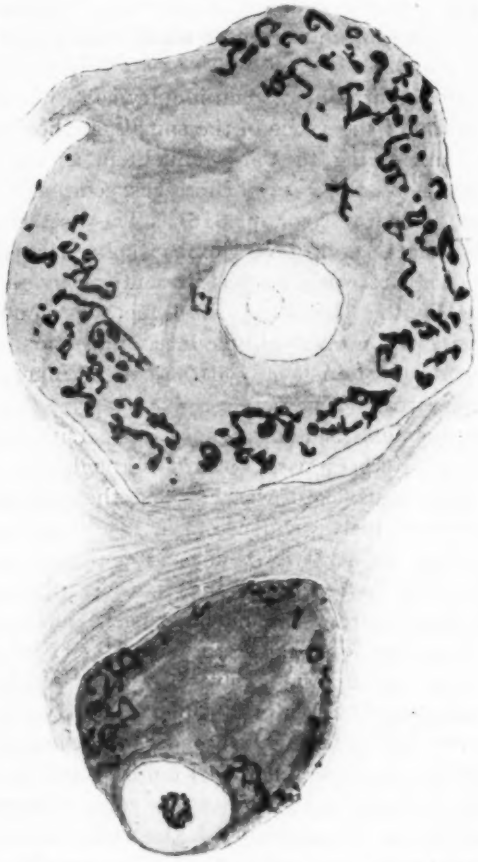

FIG. 4.—Retispersion in neurones of 7th lumbar ganglion, left side, showing the clear space about base of axone. Seven days after section of left sciatic nerve. Same experiment as that of fig. 3.

opposite side (fig. 4). Now, however, fragmentation of the apparatus had begun somewhat and also chromatolysis was present in many of the cells. On the sixteenth day chromatolysis was at its height, but retispersion seemed to have passed its apex. Although in certain cells

retisolution was more extreme than in any seen previously, there was a larger proportion of cells with a complete Golgi apparatus in its normal position than on the seventh day. In the larger cell (fig. 4) the absence of reticulum from the neighbourhood of the axone is striking. In general, that part of the cell which becomes free from all Golgi apparatus has the shape of a pear whose stem corresponds to the axone (see also fig. 3).

Interest naturally turns next to an inquiry as to whether cutting of the central extension of the axone of the spinal ganglion cell is followed by retispersion. According to Van Gehuchten, as has already been stated, this cutting of the central fibres is, curiously enough, not followed by chromatolysis ([30], p. 316). The posterior nerve roots proximal to the sacral and lower lumbar spinal ganglia were cut. Seventeen days later, retispersion was present, although not extreme. Double staining by the Cajal and Nissl methods showed the Nissl bodies for the most part normal in amount and location, although in a few cells they could be stained only at the cell periphery in the mesh of the Golgi apparatus.

The attempt was next made to produce retispersion in those sensory cells some of whose fibres pass upward through the cord without interruption. The spinal cord was cut and the ganglia lying below examined; but no conclusive evidence of a resultant change could be made out two, four, six, or twelve days after section or semisection of the cord. It must be borne in mind, however, that only a part of the central neurone-outgrowth could have been thus severed. Other divisions of the neurone end below the site of section.

Negative results.—Four and twelve days after section of the cord above, no change was found in the Golgi apparatus of the anterior horn cells. Seven days after an aseptic decerebration performed on a cat by H. C. Bazett (to whose kindness I am indebted for the material), no changes could be found in the cord motor cells. One must conclude that the alterations in the activity of the reflex arc following cord section or decerebration, are not reflected by any detectable change of the Golgi apparatus in the motor cells.

Local tetanus was produced in one hind limb of cats by opening the sheath of the common peroneal nerve and placing in and about it 30 to 60 mgm. of powdered tetanus toxin. Rigid tetanic extension of the limb followed in about three days. Six, twelve, and seventeen days later no definite change could be observed in the cord or ganglion cells corresponding to the tetanized limb.

The injection of a lethal dose of strychnine (0·83 mgrm.), once repeated, in a decapitated preparation (Sherrington [27], pp. 137-141) produced recurring tonic and clonic spasms over a period of four hours. Subsequent examination of the Golgi apparatus in the spinal cord cells revealed no changes.

If, therefore, it is possible to produce a condition of fatigue in the cells by tetanus or strychnine convulsions, at least there is no associated change apparent in the Golgi apparatus.

Summary of Results.

Decerebration and high section of the cord are without effect on the Golgi apparatus of the anterior horn cells. Following tetanus, there were no changes in the cord or ganglion cells. Administration of strychnine, causing repeated convulsions, likewise gave a negative result. The increased muscular tone of decerebrate rigidity and the muscular flaccidity following cord section must be associated with considerable variation in the activity of the motor cells. Likewise, the constant muscular contraction of tetanus, which is sufficient to cause actual muscular wasting, indicates greatly increased activity on the part of the motor cells. Nevertheless, under the above conditions, I have not been able to discover any change in the Golgi apparatus of the neurones in the spinal cord or ganglia.

Section of the axone is followed by retispersion, a specific alteration of the Golgi reticular apparatus in nerve cells. The complete reaction is made up of three stages: (1) Displacement of the unbroken apparatus to the periphery of the cell and away from the axone-hillock—retispersion (figs. 3 and 5). (2) Dissolution of the reticulum—retisolution (fig. 6); frequently there is very little evidence of retisolution. (3) Reconstruction.

In general, chromatolysis and retispersion are associated, but they may appear independently. Retispersion has been the first to appear in the experiments reported here. In a particular cell, the reticulum may be peripheral or even absent, while the nucleus remains central and the chromophil substance appears normal.

Retispersion appears early in Clarke's column, being far advanced on the fourth day after cord section. The motor cells seem to be more resistant to alteration. The Golgi apparatus appeared at the periphery of a small proportion of these cells on the seventh day and this became quite general on the sixteenth day after cutting the sciatic, but fragmentation was infrequent.

In the spinal ganglia, after section of the peripheral neurone-outgrowth, the fourth day saw the beginning and the seventh day the height of retispersion, with the appearance, also, of considerable retisolution. The cutting of the central division of the axone-outgrowth, the posterior nerve root, induced retispersion in the corresponding ganglia. But section of the cord failed to produce any change in the ganglia below, probably due to the fact that all of the central fibres could not have been thus severed.

To sum up briefly: In the above experiments, no changes were observed in the Golgi apparatus, following variations in nerve cell activity. The apparatus showed no detectable alteration after the removal of the influence of *connecting* neurones, but it exhibited a delicate and specific response, retispersion, to a lesion of its axone-outgrowth, whether central or peripheral. Retispersion is an added feature of the cellular reaction to axone interruption, usually called chromatolysis. It is ordinarily the first and in some cases the only manifestation of this cellular reaction.

In conclusion, it is a pleasure to thank Professor Sherrington for his interest and helpful criticism; also, Mr. H. M. Carleton and Dr. J. B. Gatenby for many useful suggestions.

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Section of Neurology of the Royal Society of Medicine.

President—Dr. W. Aldren Turner, C.B.

Dr. S. A. Kinnier Wilson read a paper "On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits,"[1] which appears on p. 220 of this number. A discussion followed, in which took part :—

DISCUSSION.

Dr. H. C. Bazett: I have found Dr. Kinnier Wilson's paper of great interest since his cases show such striking similarities with the decerebrate animals described by Professor Sherrington, and yet in some respects no less definite contrasts. I have recently been employed in Professor Sherrington's laboratory in developing a technique by which cats may be kept under observation for two or three weeks, even after removal of all nervous tissue lying anterior to the posterior colliculi. These chronic decerebrate animals differ only slightly from the acute preparations, but in some ways they provide a better standard for comparison with clinical material, and their importance lies in the fact that the localization of the lesion can be accurately determined.

The main similarities are that in these animals too the extensor rigidity may be maintained for days or weeks, that this position may alternate with positions of flexion, and that tonic fits with marked opisthotonos may be occasionally seen soon after the operation. A common result is to obtain an animal, which keeps up usually a position of extensor rigidity, but which after a few days also shows occasionally positions of flexion, and this flexion is sometimes flaccid but may be rigidly maintained. It is also common for a preparation, which, when approached quietly (since these animals are often very sensitive to auditory stimuli), is seen to be lying in a flexed position, to assume a position of extensor rigidity as soon as any examination is attempted. In some cases this may be induced by mere auditory stimuli.

The occurrence of tonic fits is comparatively rare, and is limited to the first few hours after the operation, except in some cats in which meningitis has developed. In the early cases the opisthotonos is extreme, the respiration is generally slow but may be fast and shallow, and occasionally with a slow respiratory rate the fits themselves have a respiratory rhythm. These animals are very different from the better preparations, in which the respiratory rate is normal, and they rarely survive long if the fits are at all severe. The operation often causes a certain amount of hæmorrhage below the tentorium into the subarachnoid space, but this hæmorrhage seems to have been

[1] At a meeting of the Section, held May 13, 1920.

particularly noticeable in the animals which have shown tonic fits. In any case the origin of these fits in the nervous system below the corpora quadrigemina is confirmed.

The main contrasts are that these preparations never show any athetoid movements or tremor, that they never show a pyrexia but a complete loss of temperature control with a rapid fall of body temperature, unless they are artificially kept warm.

They have a temporary glycosuria which disappears within about three days after the operation; they show brisk corneal reflexes, while vocalization in the form of purring, growling, and very rarely mewing may occur.

A few of the animals have survived sufficiently long to allow degeneration to take place, and the remaining parts of the central nervous system are being examined for me by Dr. W. G. Penfield, so that further data should shortly be available. It is at any rate clear that decerebrate rigidity persists even after the rubrospinal tract is mostly degenerated, so that if the red nucleus is responsible for the rigidity it must be the lower part of this nucleus that is concerned: and it is established that the rigidity is not due to the excitation of nerve fibres divided in the operation, since in this case the rigidity could not be maintained after the degeneration of the fibres.

Dr. WALSHE: While we may agree with Dr. Wilson that tonic or postural "fits" are probably of the nature of accentuations of Jackson's cerebellar attitude, I do not think that Jackson's view of the origin and relationship of these phenomena has always been correctly interpreted by those who have at various times referred to his work. In this respect, I notice that Dr. Wilson has adopted the usual view that Jackson regarded both the persisting attitude and the "fits" as identical in origin. This is not what Jackson held, and I have always been puzzled to understand how it was that in this connection he departed from his usual teaching in respect of rigidity and ascribed it in this instance to *cerebral* influx. If we refer to the papers in which he described his cases of cerebellar tumour with "tetanus-like seizures" and cerebellar attitude, which were reprinted in vol. xxix of BRAIN in 1906, we find (p. 432) the statement that "the patient's persisting attitude (cerebellar paralysis with rigidity, that is, a cerebellar attitude). . . . was, in general appearance, like that in the tetanus-like seizures, but the process of causation of the two conditions is, in my opinion, very different." And again (on p. 441), "the rigidity fixing the attitude is the result of cerebral influx, that influence being no longer antagonized, or being less antagonized, by cerebellar influence. Tetanus-like seizures are, I submit, owing to changes of instability set up in nerve cells near the tumour, owing to a cerebellar discharge lesion; the tetanus-like seizures are owing to occasional intense discharges of this persisting discharge lesion." For various reasons it is extremely difficult to believe that phenomena so closely alike in their characters can differ widely in origin, or that the persisting attitude is an expression of cerebral influx. It is far more probable that, whether we accept their identification with decerebrate rigidity or not, both have a common cause, as Dr. Wilson believes. However, it is not correct to attribute this view to Jackson.

Publications Recently Received.

Cerebrospinal Fever. By Michael Foster, M.A., M.D., and J. F. Gaskell, M.A., M.D. Pp. 222. Cambridge University Press, 1916.

In this volume the authors give a systematic account of cerebrospinal fever, based upon their extensive experience in the epidemic of 1914-15. They show reasons for preferring this term to the longer one in common use, epidemic cerebrospinal meningitis. The book deals fully with every aspect of the disease, but is admirably concise. The clinical section is particularly well arranged, and we have seldom read a clinical description at once so complete and so lucid. The chapters devoted to the evolution and course of the malady and its diagnosis are excellent and prepare the reader for the later chapters dealing with the various clinical forms of cerebrospinal fever, so that these do not have the arbitrary and artificial precision so common in clinical classifications.

The authors have attempted, as far as possible, to correlate the clinical manifestations with the underlying pathological lesions. They regard the essence of the disease as a true infection of the cerebrospinal system, causing death in fatal cases by the intensity of the pathological process, and not as a meningococcal septicæmia in which the blood-borne infection is the potent factor.

Both toxæmic and physical factors, however, play a part in the disease process. Thus, they suggest that the purulent exudate overlying the brain acts both by poisoning and by compressing the brain. It is probable, however, that some of their suggestions in this respect have but little foundation. For example, they regard the characteristic head retraction as a manifestation of increased intracranial tension. If this were so, we should expect to find head retraction a common symptom in cerebral tumour, whereas it is of extreme rarity. It is more likely that it is an expression of the extensor rigidity which Jackson described under the name of cerebellar attitude, or it might even be a reflex spasm of the neck extensors from meningeal irritation. The work of Cushing and his school upon the pathways of the cerebrospinal fluid and upon the production and nature of internal hydrocephalus was not known in this country at the time this monograph was written, and it throws light upon several points raised by Foster and Gaskell, and perforce left unanswered by them.

In the chapter devoted to prognosis they note the rarity of residual paralytic phenomena and the absence of mental defect as a sequel of cerebrospinal fever.

They hold that the essence of all treatment lies in the adequate drainage of the subarachnoid space by repeated lumbar punctures, long persisted in in chronic cases. The object of each puncture should be complete evacuation of the excess of fluid, which is always found, except in a few chronic suppurative cases.

The series of coloured plates, at the end of the text, scarcely do justice to this valuable monograph, which must become the standard work of reference on this disease in the language. There is a complete bibliography.

Instinct and the Unconscious. A Contribution to a Biological Theory of the Psycho-neuroses. By W. H. R. RIVERS, M.D., D.Sc., LL.D., F.R.S. Pp. 252. Cambridge: University Press, 1920.

After many years spent in teaching psychology and the physiology of the senses at Cambridge, Dr. Rivers turned his attention to anthropology, and is well known as the author of the "History of Melanesian Society," and for his "Studies on Kinship and Social Organization." After the outbreak of war, he was engaged in treating wounds of peripheral nerves, but soon became attached to Maghull Military Hospital, and later to Craiglockart War Hospital, where he had unrivalled opportunities for gaining experience of the psychoneuroses of war. Later, he was appointed to the staff of the Royal Air Force Hospital at Hampstead; here, and on the aerodrome at Hendon, he accumulated an unique experience of the temperament and neuroses of the flying man.

The results were embodied in a series of papers published during the last three years; these form six appendices at the end of this book, and contain an account of "A Case of Claustrophobia," "The Repression of War Experience," "War Neuroses and Military Training," and an excellent short note on "Wind-up."

The bulk of the work, however, is occupied in an attempt to bring the abnormal phenomena of mental life into harmony with the processes and activities with which we are now familiar on the physiological level. Such subjects as the unconscious, suppression and inhibition, the nature of instinct, the danger instincts and suppression, dissociation, suggestion, sleep, the substitution neuroses, regression and sublimation, are dealt with in a series of short chapters. These are clear and admirably written, and will be of the greatest value to all who are interested in the nature and workings of the unconscious. Moreover, the theory that instincts are suppressed primitive mental activities, which may be either held entirely in check or used in part, is a brilliant addition to our knowledge. Dr. Rivers has much to say on the mechanism of suppression and dissociation, which is equally luminous. It is impossible in a short space to give an adequate account of the originality and interest of this book, which deserves repeated reading.

Injuries of the Peripheral Nerves. By HENRY S. SOUTTAR, C.B.E., F.R.C.S., and EDWARD W. TWINING, M.R.C.S., L.R.C.S. Pp. 152 with 30 figures. Bristol: John Wright, 1920.

This is an admirable book clearly written and excellently illustrated. No attempt is made to enter into theoretical questions, but the gross effects produced by division of each of the larger nerve trunks are considered in detail, with especial relation to the site and nature of the injury. As might be

expected from a surgeon of Mr. Souttar's wide experience, the various operative procedures are clearly described with the help of admirable drawings. It is encouraging to find so close an agreement between the conclusions arrived at independently by the authors of this work and the Committee of the Medical Research Council. It shows that, at any rate in England, a harmonious body of opinion is gradually growing up on the diagnosis and treatment of peripheral nerve injuries. We strongly recommend this practical book to all who are interested in the problems of the surgery of nerve lesions.

The Diagnosis and Treatment of Peripheral Nerve Injuries. [Report of the Committee upon Injuries of the Nervous System.] Published by the Medical Research Council. Pp. 59. London: His Majesty's Stationery Office, 1920.

An attempt has been made in this pamphlet to lay down the general principles which should guide diagnosis and treatment in cases of injury to the peripheral nerves. But, in addition, the main nerve trunks are considered individually, and the various difficulties, due to peculiarities in their constitution and function, are considered in each instance. All theoretical questions are avoided and the advice offered, or procedure described, is given in the simplest terms.

Part I deals with nerve injuries in general, treating succinctly of diagnosis, non-operative treatment, the nature of the various operations to be performed, the signs of returning function and the phenomena of pain. In Part II, the signs and symptoms of injury, together with the methods of treatment to be adopted, are considered for each of the main nerve trunks in turn. In Part III the histological appearances are described as they appear under different conditions of injury. Finally, the treatment of painful states, such as "causalgia," and the factors which produce them are dealt with briefly. The Report is intended as a succinct guide for those who are engaged in treating nerve injuries resulting from the war, or in assessing the various degrees of disability they may produce.

Diagnosis and Treatment of Brain Injuries with and without a Fracture of the Skull. By WILLIAM SHARPE, M.D., Professor of Neurologic Surgery, New York Polyclinic Medical School. Pp. 757. Philadelphia and London: Lippincott, 1920.

The author deals with methods of diagnosis and describes in detail the signs and symptoms, treatment and progress of individual patients with various forms of brain injury. He rightly insists on the importance of considering the form and situation of the lesion of the brain, as opposed to the injury of the skull. For, in treating patients with head injuries, the important factor is not

the presence or absence of a fracture, but rather the nature of the intracranial damage and increased cerebral tension. Stress is also laid on the necessity for considering the future condition of the patient, as well as preservation of his life.

The book is divided into three main parts:—

(1) General conditions, such as pathology, diagnosis and treatment, including operative technique; (2) Brain injuries in adults, both acute and chronic; (3) Brain injuries in new-born babies and children. Full records are given of 195 cases and the illustrations are good.

It is urged that the enormous mortality, often 50 per cent., following operative treatment in the acute stage, is largely due to the inopportune time chosen by surgeons for interference. The two periods during which operation is distinctly contra-indicated are, firstly, during the early stage after the injury, when the patient is suffering from shock, and, secondly, after signs have appeared of medullary compression. In the author's opinion, expectant palliative treatment is successful in two-thirds of the cases, excluding depressed fracture of the vault; but in the remaining third, where there is considerable increase of intracranial pressure, the skull and dura should be opened, preferably in the subtemporal region. We note with surprise that all methods for closing cranial defects are condemned, and no mention is made of the distressing symptoms which are liable to develop in patients with large openings in the skull.

The Medical Problems of Flying, including Reports I to VII of the Air Medical Investigation Committee. Published by the Medical Research Council. Pp. 272. London: His Majesty's Stationery Office, 1920.

During the course of the war, flying was gradually carried on at greater and greater heights, so that by the autumn of 1916 "mountain sickness" in various degrees gravely increased the difficulty and dangers met by flying officers in the course of their work in the air. Much experimental work was undertaken by individual medical officers, expert physiologists, and civilian physicians, and a series of special reports were published under the ægis of the Medical Research Committee. The papers which comprise this volume contain much that is both directly and indirectly of interest to neurologists. They deal with the reaction of pilots and observers to diminish oxygen pressure, as measured by Professor Dreyer's method, and by the various tests introduced by Colonel Flack. The papers on "Methods used in selecting Candidates for Flying," "On the Sense of Stability and Balance in the Air," on "Mental Aptitude for Aviation," and on "Reaction Time Tests carried out on Flight Cadets," all bear on the functions of the central nervous system in an unexpected and novel manner. Some of the tests described are worthy of more extended application to neurological practice.

Les Maladies nerveuses. Cours professé à l'Université de Louvain. By the late Professor A. VAN GEHUCHTEN. Pp. 645. Louvain: Librairie Universitaire, 1920.

Professor van Gehuchten was preparing to publish the course of lectures delivered by him in the University of Louvain, when the work was stopped by the outbreak of war. The first four hundred pages were already in print, the remainder was in manuscript. The plates prepared to illustrate this book were burned by the Germans, but they have been excellently reproduced from photographs, and a large number of diagrams show the anatomical relations necessary for understanding the signs of the various diseases described. The whole has been passed through the press by the filial piety of Dr. Paul van Gehuchten in memory of his distinguished father.

As the title indicates, this book is intended for students, not for neurologists, and probably for this reason all references are omitted. Organic diseases are classed under such headings as peripheral nerves, muscles, spinal cord, mid-brain, &c. This necessitates somewhat summary treatment, for all morbid conditions are described in terms of anatomy, and little attention is paid to disturbance of function. The last section of the book is devoted to "functional nervous disorders" grouped under the headings of chorea, paralysis agitans, epilepsy, hysteria, neurasthenia and traumatic neuroses. It is a profound misfortune that the lamented author died before his work was completed, and without having the opportunity to embody the great advances in neurology made during the last six years.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.